Seventeenth Annual Meeting — American College of Chest Physicians
*Ambassador Hotel, Atlantic City, New Jersey, June 7-10, 1951*

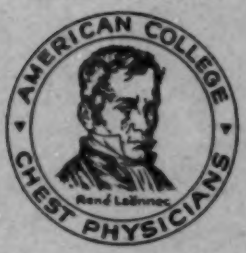
VOLUME XIX NUMBER 3

# DISEASES

## *of the*

# CHEST

OFFICIAL PUBLICATION

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1. Krasno, L., Karp, M., and Rhoads, P. (1948), The Inhalation of Penicillin Dust, J. Amer. Med. Assn., 138:344, October 2.
2. Krasno, L., and Rhoads, P. (1949), The Inhalation of Penicillin Dust; Its Proper Role in the Management of Respiratory Infections, Amer. Proc., 11:649, July.

# DISEASES *of the* CHEST

OFFICIAL PUBLICATION

OF THE

AMERICAN COLLEGE OF CHEST PHYSICIANS





EXECUTIVE OFFICE
500 North Dearborn Street, Chicago 10, Illinois
MURRAY KORNFELD, *Managing Editor*

VOLUME XIX MARCH 1951 NUMBER 3

# CONTENTS:



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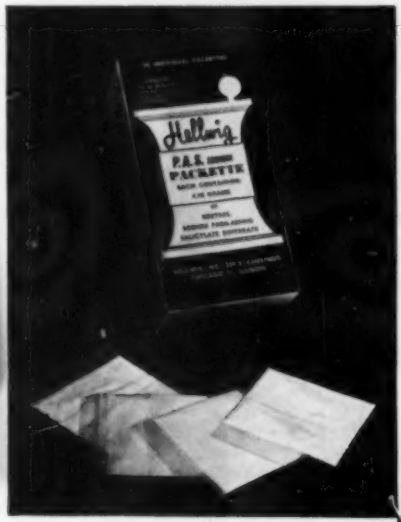


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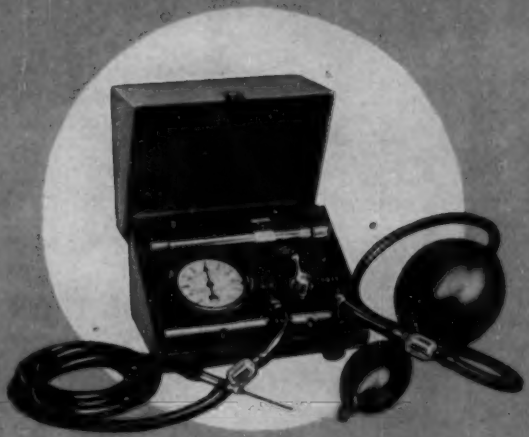

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DISEASES *of the* CHEST

VOLUME XIX MARCH 1951 NUMBER 3

# Early Diagnosis and Treatment in Tuberculosis Prophylaxis

MANOEL de ABREU, M.D., F.C.C.P.*
Rio de Janeiro, Brazil

## *History of an Idea*

Our early experiments on indirect radiocinematography, in 1918, and our experience in thoracic radioscopy, at the Laennec Hospital in Paris, are perhaps the origin of the idea here presented. In 1923 and 1924 we made our first fluorographic researches in Rio, the aim of which was the mass examination. In 1926 the situation was as follows: no researches on fluorography were carried on anywhere; the fluorographic apparatus did not exist; nobody expected the employment of this method; there was not a social mentality, in the radiologic field.

We resumed, in 1935, our interrupted researches and in 1936 we published about the new method. The following were the most important facts: we showed that fluorography enabled the diagnosis of chest diseases; we used special apparatus for collective fluorography; finally, we founded the first Fluorographic Center, whose influence was decisive in the advent of social radiology.

In 1936 and '37 the apparatus built in Rio, already enabled the mass x-ray examination; three Centers, at the Public Health Department, at the Navy (Rio) and at the Public Health Department (Vitoria City), performed the thoracic control of several social groups. Our book "Fluorographic Survey, Livraria O Globo," 1937, contains the early experience about the method.

The first model of the apparatus had vertical columns, where the pyramidal trunk and the tube slid by means of counterweights. The special camera had 10-meter film reels. We started in 1936 on the 60 mm. film, that was replaced by the 35 mm. size, which is to be preferred for examining on a large scale.

*Professor of Radiology, Medical Faculty, University of Rio de Janeiro, Brazil.
Presented before the 16th Annual Meeting of the American College of Chest Physicians, San Francisco, California, June 24, 1950.

At present, we have in Brazil, about 200 Fluorographic Centers. The most important problem is not the apparatus that works faster; although valuable, the technical conditions are not so important as the social employment of the method: choice of strategic points, fixed and mobile units, sanitary propaganda, well prepared technicians, new methods of individual diagnosis, perfect understanding of the final purposes of the periodic x-ray examination.

### *Shadow and Lesion: Radiogeometry*

Between the years 1924 and 1928 we published many works on the principles of the image formation. The sharpness is always caused by the tangent or the parallel incidence of the rays on the opposition surfaces. Later, we applied the same laws in tomography (1940). Generally, the visibility of a lesion does not depend on the dimensions of the film, but on the direction of the rays through the surfaces between two zones of different absorption.

The idea and the performing of mass fluorography were in part inspired by the knowledge of radiogeometry. Although it was published in France in 1926 and 1930, and in Germany in 1935, the theorems of radiogeometry are almost completely unknown. We will soon publish a book about the principles of radiologic interpretation.

### *Collective and Individual Examinations*

The collective control separates the abnormal cases; the individual examination (clinical, radiologic, bacteriologic, tuberculinic, anatomo-pathologic, etc.) performs the diagnosis, that is, establishes the etiology, the pathology and the evolutive potential. They are quite different. Mass fluorography aims only to show the abnormal individual, the bearers of pulmonary shadows or mediastinal modifications. On the contrary, the individual x-ray examination presents a great complexity. We think that teleradiography has generally the same value as fluorography. The individual chest radiology requires, according to the cases, several incidences of tangential direction, numerous tomographies, special techniques (kymography, bronchography, angiocardiography, etc.).

At the direction of Pulmonary Diagnosis Service, since 1944, we have claimed for larger and equipped laboratories, on account of the great number of bearers of shadows, shown by the fluorographic survey.

We employ two new individual methods, *pulmonary or bronchial lavage* (1944), which has replaced gastric lavage, and the *simultaneous tomographies* (1947), systematically employed in cases of apical lesions. Simultaneous technique performs three sections during a single film-tube motion.

### *Apparently Healthy and Apparently Sick*

The periodic fluorographic survey (yearly), including all the urban and rural population, shows the bearers of pulmonary or mediastinal abnormalities. Among the apparently healthy people are the unapparent sick ones, on whom the disease generally begins. Certain groups very much exposed and individuals who live with contagious people must be examined two or three times a year.

However, the periodic survey is not sufficient, because there are many diseases of rapid evolution: acute and sub-acute infections, neoplasms, etc. Then, the apparently sick, those who present signs of a disease, still undetermined, those who miss their work feeling ill, must be fluorographed at the first symptoms.

### *Collective Fluorographic Centers*

At present Mass Fluorography begins to be understood and applied in all its collective amplitude. However, during 10 years the employment of the method was not intensive enough. The world was not prepared to perform the real social radiology. My first apparatus was inaugurated in 1936. The first Fluorographic Center of the Health Department in Rio, has been in use since March 1937. Two other Centers started in the same year, in Rio and Victória. Between 1938 and 1945 many Fluorographic Centers in Brazil, especially in Rio and Sao Paulo, were in condition to make millions of chest examinations. But, they were nearly paralyzed. The old conception of tuberculosis prophylaxis, based on hospital isolation, has prevailed and is still prevailing. The diagnosis without hospital isolation was considered useless. No solution could be found, because the mortality was very high and the economic conditions were relatively low. It would be impossible to build 150,000 beds and to maintain them. I have been affirming with obstinacy since 1936 that the most important prophylactic factor is the *early diagnosis and the early treatment*. I wrote in my first paper about the subject: *the periodic examination of the whole population is the only way to detect all cases of early tuberculosis* (Medical Society, Rio, August 1936).

On account of the mentioned misunderstanding, the fluorographic surveys development was very slow, as follow:

| | |
|---|---|
| 1937 | 20,000 |
| 1938 | 30,000 |
| 1939 | 50,000 |
| 1940 | 50,000 |
| 1941 | 60,000 |
| 1942 | 70,000 |
| 1943 | 80,000 |
| 1944 | 100,000 |

Fortunately, since 1945, the Previdence Institutes have started large fluorographic surveys. Otherwise, the number of specialized physicians greatly increased. The following numbers indicate the recent fluorographic development:

| | |
|---|---|
| 1945 | 150,000 |
| 1946 | 200,000 |
| 1947 | 500,000 |
| 1948 | 1,200,000 |
| 1949 | 1,500,000 |

In the United States the same delay has been observed. The mass fluorography was introduced in 1939 by D. O. N. Lindberg and I. Seth Hirsch, who visited the Fluorographic Center in Rio at the end of 1937. I was in the United States in 1940, during the Pan-american Congress and exposed the advantages of the fluorographic survey of the whole population; but it was yet too early. Only in 1945 the new method began to reach a large social amplitude. Here are the recently published figures:

| | |
|---|---|
| 1945 | 1,000,000 (army) |
| 1948 | 12,000,000 |
| 1949 | 14,000,000 |

We believe that the x-ray social control does not belong to the dispensaries. They are different institutions. The social radiology do not examine tuberculous people, but all of the population, where the majority is of normal individuals and where there are patients of different diseases. So, the bearer of chest diseases, detected by the Fluorographic Center, are sent to specialized clinical services, among which are the anti-tuberculous dispensaries.

### *Social Prophylaxis and Mass Fluorography of the Chest*

Morbidity and mortality vary in accordance with the epidemiological factors and the presence of open tuberculosis cases without medical or prophylactic control. After the last 14 years of survey experience, we think that tuberculosis will certainly be overcome.

At present the most important factor of prophylaxis is the *early treatment*, in accordance with the *early diagnosis*.

In Brazil, before the plain development of the fluorographic survey, the epidemic increased progressively, in accordance with several causes: urban concentration, industrialization, rural people mobilization and other social and epidemic factors.

The following numbers explain the situation in Rio and Sao Paulo before the plain development of fluorographic surveys:

*Tuberculosis Mortality Rates**

| | 1930 | 1935 | 1940 | 1945 |
|---|---|---|---|---|
| S. Paulo | 124 | 130 | 135 | 145 |
| Rio | 308 | 309 | 327 | 342 |

*Per 100,000.

In 1937 we had the first three Mass Fluorographic Centers. At present, we have many fluorographic units, most of them, about 100, concentrated in Rio and Sao Paulo (cities of 2,000,000 and 1,700,000 respectively). The units are numerous, but do not produce enough, in view of difficulties which were met in developing new tuberculosis prophylaxis. However, in the last few years, the survey activity improved very much especially in Rio and Sao Paulo.

The rapid decrease of tuberculosis mortality results in progressive use of the fluorography that enables the early diagnosis and the early treatment. Here are the mortality rates in Rio, during the last five years:

*Tuberculosis Death Rates in Rio*

| | 1945 | 1946 | 1947 | 1948 | 1949 |
|---|---|---|---|---|---|
| Number of Deaths | 6,556 | 6,267 | 6,509 | 5,789 | 4,930 |
| Mortality Rate* | 342 | 326 | 325 | 289 | 242 |

*Death Rates by Age in Rio*

| AGE | 1945 | 1946 | 1947 | 1948 |
|---|---|---|---|---|
| 0- 1 | 46 | 59 | 51 | 65 |
| 1- 2 | 64 | 95 | 94 | 104 |
| 2- 4 | 107 | 94 | 137 | 124 |
| 5- 9 | 93 | 76 | 68 | 64 |
| 10-14 | 118 | 110 | 96 | 91 |
| 15-19 | 675 | 647 | 654 | 538 |
| 20-29 | 2,022 | 1,886 | 1,998 | 1,738 |
| 30-39 | 1,526 | 1,484 | 1,445 | 1,289 |
| 40-49 | 974 | 967 | 1,014 | 898 |
| 50-59 | 602 | 533 | 591 | 511 |
| 60-+ | 324 | 314 | 351 | 364 |

*Per 100,000.

The mortality decrease is very important among the adults, especially between the age of 20 and 40 years. These groups composed of employees and workers are more frequently examined in the fluorographic surveys. Among children the case reverses, the mortality increases during the first four years of life.

We must say that the social conditions are not better; we believe that they are worse. The number of hospital beds is also about the same (2,000). The only conditions that have changed, in the last few years, were the intense fluorographic survey and the use of antibiotics.

It must be noticed that the progressive mortality among children does not show the influence of the large and progressive use of BCG. The problem is a complex one, and we do not know how to explain this apparent discordance.

In Sao Paulo the decrease of mortality is not so expressive:

*Tuberculosis Death Rates* in Sao Paulo*

| 1945 | 1946 | 1947 | 1948 |
|---|---|---|---|
| 145 | 134 | 135 | 117 |

*Per 100,000.

The above rates do not represent the real situation, because the tuberculous people of the interior come to the city to get treatment and hospitalization. It seems that here is a very clear decrease of mortality of the city residents.

We think that the epidemic declination already evident, is only beginning: the mass surveys have made remarkable progress in the last few years, performed by the Tuberculosis Departments and Insurance Institutes.

When we presented the mass fluorography, 14 years ago (1936), we were convinced that the cases of hidden tuberculosis were the principal cause of the high mortality rates. The open tuberculosis without medical and prophylactic control would disappear with periodic large x-ray surveys.

The present results confirm our early previsions. *We believe that precocious diagnosis and precocious treatment are the most important factors in tuberculosis prophylaxis.*

# The Surgical Treatment of Circumscribed Intrathoracic Lesions:

## Lesions Found on Routine Thoracic Roentgenologic Examinations, with Absence of Subjective Symptoms*

STUART W. HARRINGTON, M.D.†
Rochester, Minnesota

The primary purpose of this paper is to point out the importance of establishing a definite diagnosis of all abnormal lesions found in the thorax by roentgenologic studies, regardless of whether or not the patient presents any subjective symptoms. I shall also point out some of the different kinds of circumscribed lesions I have found by exploratory thoracotomy in patients who have had no thoracic complaint and whose lesions were first made known by examination of routine roentgenograms of the thorax.

The finding of circumscribed lesions by routine roentgenologic examination of the thorax in patients who do not have any subjective symptoms presents many interesting problems in diagnosis and treatment.

I have been interested in this group of patients for a number of years. Two of the first group of five patients who had intrathoracic tumors which I removed surgically and reported in 1927 were of this type. One was a woman 31 years old, who came to the clinic in February, 1926, because of a large, rounded, circumscribed tumor occupying almost the entire upper and right part of the thoracic cavity. The presence of this tumor had been known for more than four years. It had been found elsewhere by routine physical and roentgenologic examination of the thorax. At that time the patient had no subjective symptoms. At the time of her examination at the clinic a diagnosis of malignant tumor was made and operation was advised. The tumor was removed on February 9, 1926; microscopic examination showed it to be a neurofibrosarcoma. In view of the fact that this tumor was known to be present for a number of years without causing symptoms, and then later did cause symptoms, I believe the lesion was primarily benign and then underwent malignant change.

The other patient in this group of five reported on in 1927 came to the clinic in September, 1926, for a general examination because of abdominal complaint and presented no symptoms of intra-

*Presented at the 16th Annual Meeting of the American College of Chest Physicians, San Francisco, California, June 25, 1950.
†Division of Surgery, Mayo Clinic, Rochester, Minnesota.

thoracic disease. Routine roentgenologic examination of the thorax disclosed a large circumscribed tumor in the upper and right parts of the posterior thorax. The roentgenologic interpretation was that of a malignant tumor. This patient was operated upon on September 25, 1926, at which time the tumor was completely removed by posterior extrapleural thoracotomy. Microscopic examination of the tumor showed it to be a benign neurofibroma.

The observations in these two cases, particularly in view of the fact that one of the lesions was malignant, stimulated my interest in the operative treatment of such tumors. The many different types of lesions found subsequently, as well as the relatively high percentage of malignant processes discovered, emphasize the importance of careful consideration of all such lesions.

The occurrence of intrathoracic tumors of course has been known for many years. Before the advent of roentgenologic methods of diagnosis the presence of these tumors was determined by the subjective symptoms and by physical examination of the thorax. In most instances, it was impossible to determine the type of lesion, whether the tumor originated from the tissue of the lung or from the structures in the mediastinal spaces outside the lung. During this period the diagnosis usually was obtained so late in the course of the disease that conservative methods of treatment had to be employed, because surgical treatment was considered a very hazardous procedure, was seldom used and was rarely successful. Studies made at that time to determine the character of these tumors was based upon inspection of long-standing lesions and necropsy material, and it was thought that most of these tumors were malignant.

The use of roentgen rays in diagnosis has made it possible to recognize these lesions early, before they have made serious inroads on the patient's general condition. The lack of response of most of these tumors to conservative treatment has encouraged surgical intervention with the objective of complete removal. Concomitantly, the marked advancement in methods of surgical technic has made operative removal of these lesions relatively safe. Study of these early lesions has given a different conception than was formerly held of the type of tumor found in the thorax and of the prognosis. Microscopic study of these early lesions, for instance, has shown that a large percentage of them are benign.

### *The Factor of Malignancy*

Nonetheless, all intrathoracic tumors should be considered to be potentially malignant. Tumors that remain benign often attain enormous size; they may even cause death from mechanical pressure on the important structures in the thoracic cavity which

are adjacent to the tumor. These structures either control, or are closely associated with, respiration, circulation of arterial and venous blood and lymph, deglutition as well as the functional innervation of organs lying outside the thorax.

Because of the potential malignant character of these tumors and their close proximity to the important structures contained within the thorax, it is of paramount importance that such lesions be recognized early, and that treatment be instituted immediately before a tumor has caused serious and permanent injury from pressure to the vital thoracic structures or has undergone malignant change. In primary malignant tumors, early diagnosis and operation are essential because of the greater possibility of complete removal and less likelihood of distant metastasis if they can be attacked soon enough.

### *Symptoms, If Present*

Inasmuch as this paper deals essentially with lesions which are not associated with subjective symptoms, I shall not discuss the symptoms associated with intrathoracic lesions. In a general way, however, it may be stated that the symptoms which are produced by these lesions depend greatly on the location of the tumor in the thorax as well as the type of tumor. I should also like to point out that if symptoms are present, pain will be very important,

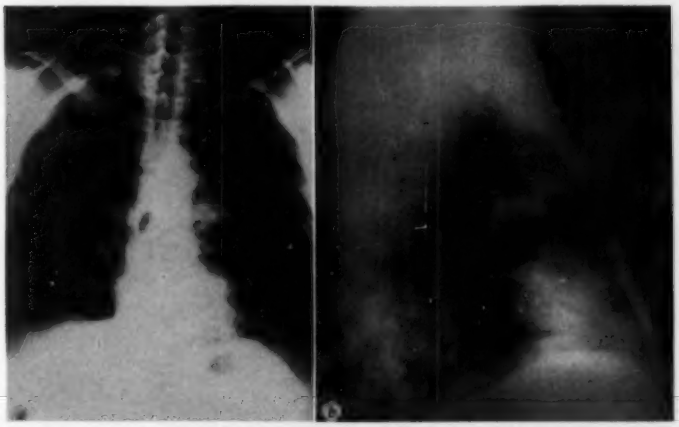

FIGURE 1a FIGURE 1b

*Figure 1a and 1b:* Anterior mediastinal teratoma containing adenocarcinoma, grade 3, squamous epithelium, various types of glands, fat, smooth muscle, cartilage, pancreas, thymic tissue, nerve tissue, glial tissue and hair. The patient was a man 38 years old. The tumor was found at routine roentgenologic examination and had produced no symptoms. The malignant teratoma was completely removed by left posterolateral transpleural thoracotomy.

not only because it helps to determine the presence of a lesion but also because it suggests the type of lesion; that is, whether it is benign or malignant. As a rule, benign lesions may attain very large size without producing pain, but if they should become malignant, they generally will produce pain. Malignant lesions, even small ones, usually are associated with pain, and are more likely to present symptoms of involvement of nerves, such as Horner's eye syndrome or paralyzed vocal cord. Benign tumors may affect nerves to produce conditions such as paralysis of the vocal cord (neurofibroma of the vagus and recurrent laryngeal nerves) or invasion into the spinal cord (neurofibroma) as well as Horner's eye syndrome (ganglionoma). Benign tumors such as these, which produce nerve paralysis, generally are not associated with pain. In fact, benign tumors may attain huge size in certain locations of the thoracic cavity, especially the posterior mediastinal region, without producing distress other than that of pressure upon surrounding structures such as the lungs, causing dyspnea, or such as the esophagus, causing dysphagia.

### *Early Recognition*

It is well known that for the past 25 years or more the medical profession has been alert to the importance of early recognition to the successful treatment of intrathoracic tumors. Complete

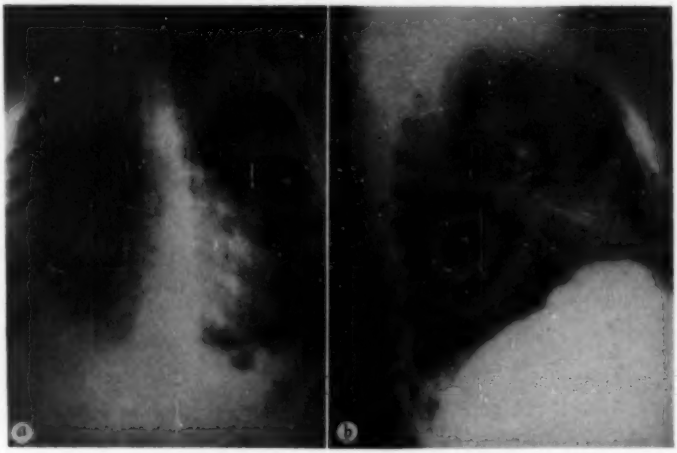

FIGURE 2a FIGURE 2b

*Figure 2a and 2b:* Cellular fibroma of the pericardium of a man 46 years old. The tumor was found at routine roentgenologic examination, and had produced no subjective symptoms. The fibroma was completely removed by left posterolateral transpleural thoracotomy.

roentgenologic and other types of thoracic examination are carried out for patients who seek medical attention for some definite thoracic complaint. It has only been in the last few years, however, that routine roentgenologic examinations have been conducted among mass groups of people who have no subjective symptoms of thoracic disease. These surveys have been carried out because it has been found that many intrathoracic lesions do not produce any definite symptoms early in the course of the disease, obviously a period in which they can be treated most effectively. In a routine roentgenogram, when the finding is indicative of a tumor, it is important that all methods of thoracic diagnosis be utilized to establish a definite diagnosis, because in many locations different types of early malignant disease may not produce subjective symptoms. If sufficient time is permitted to elapse for these tumors to produce symptoms, much valuable time will have been lost before surgical treatment is instituted and the possibility of complete eradication of the disease may be greatly reduced. The absence of subjective symptoms may be interpreted as indicating a harmless lesion, and thereby give a false sense of security or may suggest treatment by conservative measures and keeping the lesion under observation.

### *Roentgenologic Factors*

It must be kept in mind that many different types of tumor in various locations in the lungs and mediastinum have similar roentgenologic manifestations, and a definite diagnosis cannot be established even after utilization of all the different roentgenologic

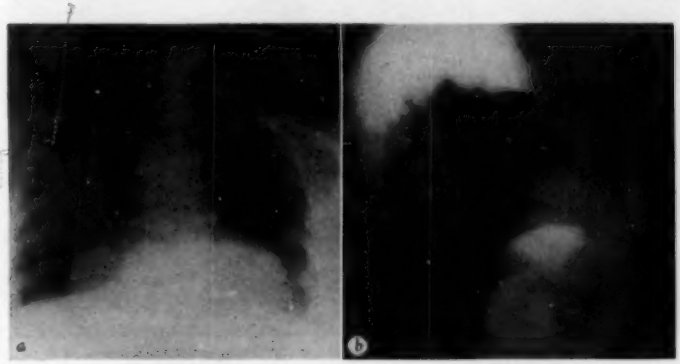

FIGURE 3a FIGURE 3b

*Figure 3a and 3b:* Coelomic cyst of the right side of the anterior mediastinum of a woman 56 years old. The tumor was found at routine roentgenologic examination, and had produced no subjective symptoms. The cystic tumor was completely removed by right posterolateral transpleural thoracotomy.

means, such as roentgenoscopy, angiocardiography and examination of the esophagus and stomach after the patient has ingested barium, to rule out any possibility of a lesion of, or impingement upon, these structures. In many instances, after utilization of all other methods of thoracic diagnosis, such as bronchoscopy, thoracoscopy, esophagoscopy, examination of sputum for malignant cells or organisms and so on, a definite diagnosis cannot be established.

### *Anatomic Situations*

*Anterior Thorax:* From a study of the types of tumors in cases of proved lesions, it has been found that the position of the tumor in the thoracic cavity is of great value in suggesting its type, for certain types of lesions are more likely to occur in certain locations than in others. In a general way, it may be stated that the most common types of tumors in the anterior thorax are the various teratoid neoplasms (mesodermal, entodermal and ectodermal [dermoid cysts]), but such tumors occasionally are also found in the posterior mediastinum. Other lesions which may be found in the anterior thorax are thymic tumors, cystic tumors, peripheral pulmonary tumors, lipomas, fibromas, thoracic thyroid and various types of connective-tissue tumors (Figs. 1, 2 and 3).

*Posterior Thorax:* The most common tumors located in the pos-

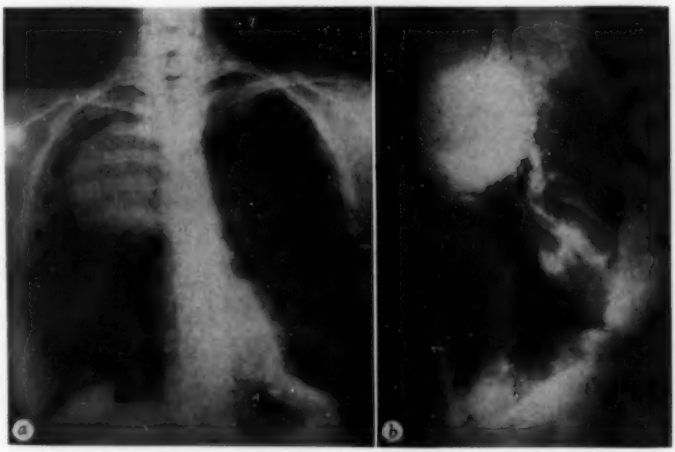


FIGURE 4a FIGURE 4b

*Figure 4a and 4b:* Neurofibroma of the right and upper parts of the posterior thorax of a woman 46 years old. The tumor was found at routine roentgenologic examination, and had produced no subjective symptoms. The neurofibroma was completely removed by right posterolateral extrapleural thoracotomy.

terior thorax are perineural fibroblastomas or neurofibromas, although these tumors also may be located along the course of the nerves along the lateral wall of the thorax, as well as in the spinal column. Other lesions found in this region are granulomatous lesions of the mediastinal lymph nodes or parenchyma of the lung, tumors of the esophagus, peripheral pulmonary tumors and cysts (Figs. 4, 5 and 6).

*Middle Thorax:* The more common lesions in the middle thorax are those of the great vessels, intrapulmonary lesions, cardiac lesions, aneurysms and lymphoblastomas. Lesions in this location require the greatest care in distinction from a surgical standpoint, because many lesions in this region are not amenable to surgical treatment (Figs. 7, 8 and 9).

*Superior Thorax:* Tumors of the superior thorax often present difficulty in distinction because in many instances they fill the entire space, so that it is difficult to evaluate the surrounding visceral relationship. The most common tumors located in this region are thymomas, intrathoracic goiters, neuroblastomas, aneurysms, cystic lymphangiomas, as well as cysts of the lung, and particularly cystic azygos lobe (Figs. 10 and 11).

*Lower Thorax:* In the lower thorax above the diaphragm, the most common lesions found are cystic or solid tumors associated

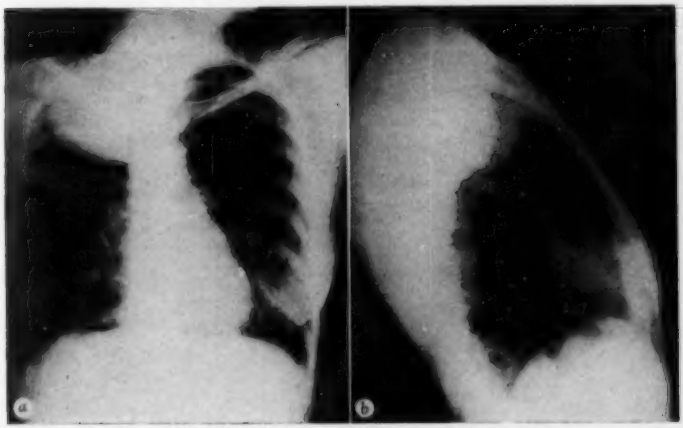

FIGURE 5a FIGURE 5b

*Figure 5a and 5b:* Neurofibrosarcoma of the upper and right parts of the posterior thorax of a woman 31 years old. This had been first found, elsewhere, four years previous to the patient's admission, by routine roentgenologic examination of the thorax. There were subjective symptoms. At the time of operation here, the patient had severe pain in the upper part of the thorax and Horner's eye syndrome. The neurofibrosarcoma was completely removed by right anterior transpleural thoracotomy.

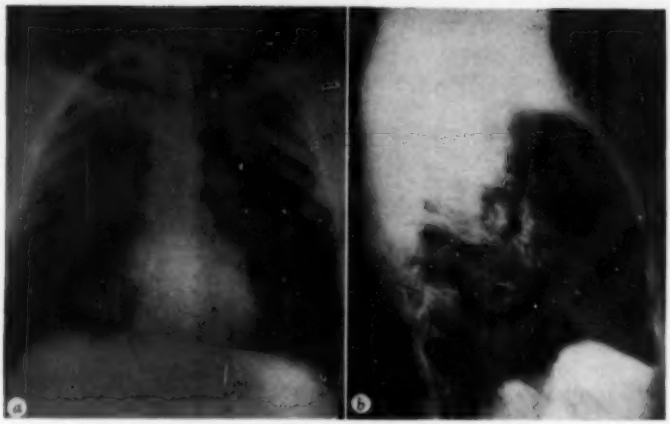

FIGURE 6a FIGURE 6b

*Figure 6a and 6b:* Dumbbell type of neurofibroma (arrows) of the posterior part of right thorax arising from the ninth thoracic nerve root of a man 27 years old. The tumor was found at routine roentgenologic examination of the thorax; the patient had no subjective symptoms. The neurofibroma was removed, five years after it had been first noted at roentgenologic examination, by right posterolateral transpleural thoracotomy.

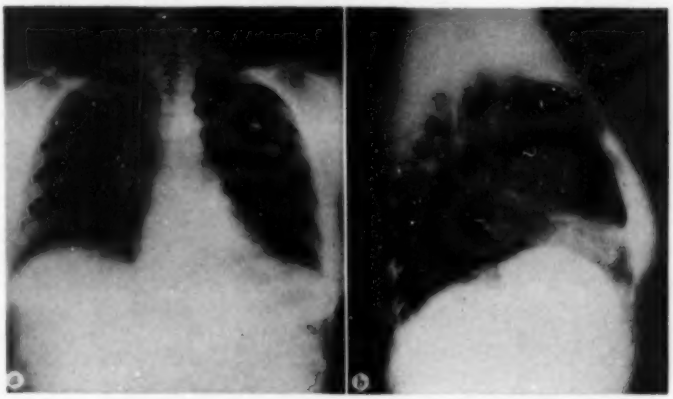

FIGURE 7a FIGURE 7b

*Figure 7a and 7b:* Chronic granuloma (arrows) of the lower lobe of the left lung of a woman 41 years old. The tumor was found at roentgenologic examination of the thorax; the patient had presented no subjective symptoms. This chronic granuloma was completely removed by segmental resection of the left lung by posterolateral transpleural thoracotomy.

with the pericardium, tumors or diverticula of the lower part of the esophagus and lesions of the diaphragm which may simulate an intrathoracic tumor such as subcostosternal hernias (foramen of Morgagni) containing omentum only or hernias of the liver through the dome of the right side of the diaphragm (Figs. 12, 13 and 14).

### *Exploratory Thoracotomy and Surgical Treatment*

In cases in which the roentgenologic findings are those of a circumscribed tumor, and in which a definite clinical diagnosis cannot be established by the use of all of our present diagnostic methods, I believe that exploratory thoracotomy, with removal of tissue for biopsy, should be carried out regardless of the absence of subjective symptoms, unless operative treatment is contraindicated for other reasons relative to the patient's general condition.

Exploratory thoracotomy for the establishment of diagnosis and removal of the lesion, if indicated, is justifiable because (1) the risk to the patient is very slight, as a result of marked improvement in technical methods as well as in preoperative and postoperative measure, (2) the lesion may be malignant and may metastasize, (3) the tumor may be undergoing malignant change, and (4) if removal of the lesion is delayed until after it has increased in size and impinges upon or interferes with the function

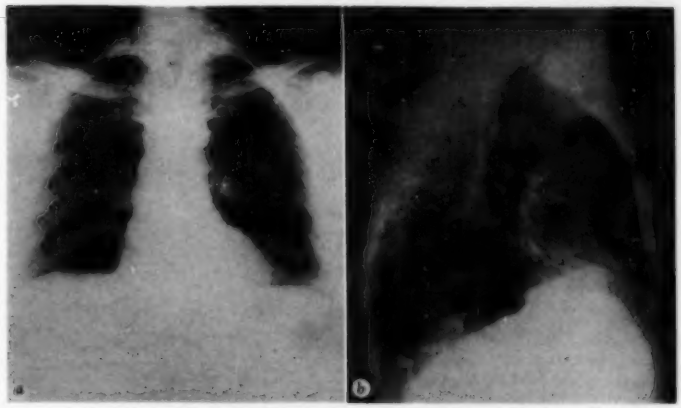

FIGURE 8a FIGURE 8b

*Figure 8a and 8b:* Hamartoma (arrows) of the upper lobe of the left lung of a man 49 years old. The tumor was found at routine roentgenologic examination of the thorax; it had caused no subjective symptoms. The hamartoma was completely removed by local excision and reconstruction of the lung through left posterolateral transpleural thoracotomy.

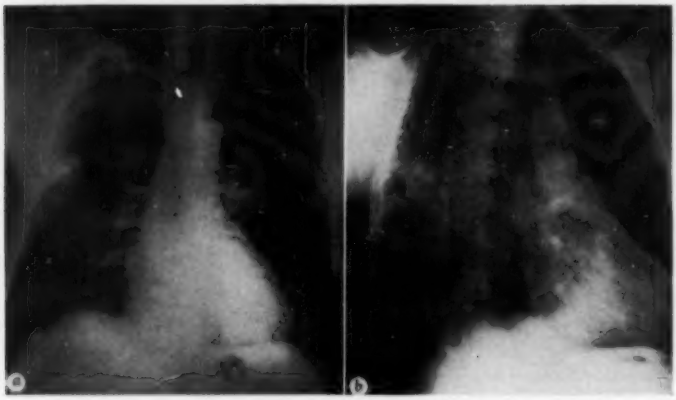

FIGURE 9a FIGURE 9b

*Figure 9a and 9b:* Peripheral carcinoma (arrows) of the lower lobe of the right lung, without metastasis to hilar nodes, in a man 58 years old. The patient had coronary disease. The tumor was found at routine roentgenologic examination of the thorax, and had caused no subjective symptoms. The peripheral carcinoma was removed by upper right lobectomy, with dissection of hilar nodes, through right posterolateral transpleural thoracotomy.

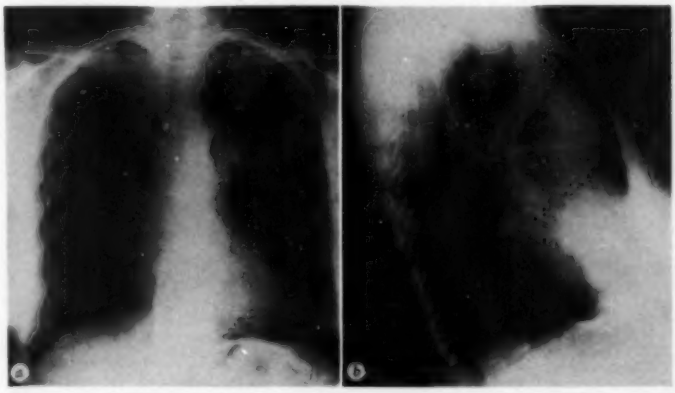

FIGURE 10a FIGURE 10b

*Figure 10a and 10b:* Thymoma of the upper part of the anterior mediastinum of a woman 64 years old. The tumor was found at routine roentgenologic examination of the thorax; the patient had presented no subjective symptoms. The thymoma was completely removed by left posterolateral transpleural thoracotomy.

of other important intrathoracic organs and structures, will be greatly increased.

Each lesion must be dealt with individually, depending upon the special findings in that particular case. In a general way, it may be stated that all lesions, whether extrapulmonary or intrapulmonary, should be completely removed in a one-stage operation. In those cases in which an exploratory operation is required to establish a diagnosis, the lesion should be completely removed at exploration, if it is found to be surgically removable. If a lesion is benign, whether the location is extrapulmonary or intrapulmonary, the operative procedure should be as conservative as possible. A benign extrapulmonary lesion should be completely removed, and any damage to surrounding structures upon which it impinges should be repaired. A benign intrapulmonary lesion, such as a hamartoma or a chronic granuloma, often can be excised from the parenchyma of the lung and the injury to the lung can be repaired by suture. This, however, will depend on the position of the tumor in the parenchyma of the lung. Safe and adequate excision of some of the larger tumors, or those centrally located close to the bronchi, may require lobectomy or, occasionally, pneumonectomy.

When a lesion is malignant the operation should always be as extensive as is necessary for adequate removal of the neoplasm

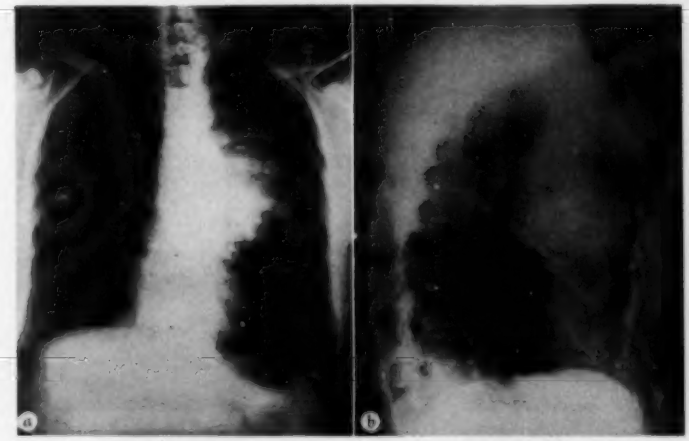


FIGURE 11a FIGURE 11b

*Figure 11a and 11b:* Intrathoracic adenomatous goiter in a woman 51 years old. The tumor was found at routine roentgenologic examination of the thorax; the patient had presented no subjective symptoms. The intrathoracic goiter was completely removed by left posterolateral transpleural thoracotomy.

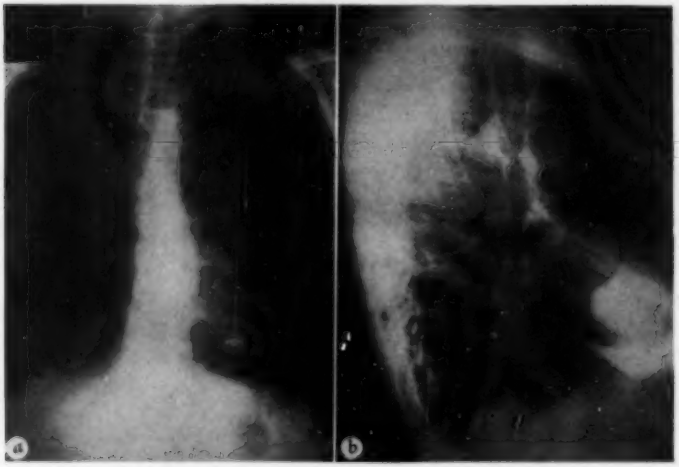

FIGURE 12a FIGURE 12b

*Figure 12a and 12b:* Pericardial coelomic cyst of the right and lower parts of the anterior thorax of a man 29 years old. The tumor was found at routine roentgenologic examination of the thorax; it had not caused subjective symptoms. The cystic tumor was removed by right posterolateral transpleural thoracotomy.

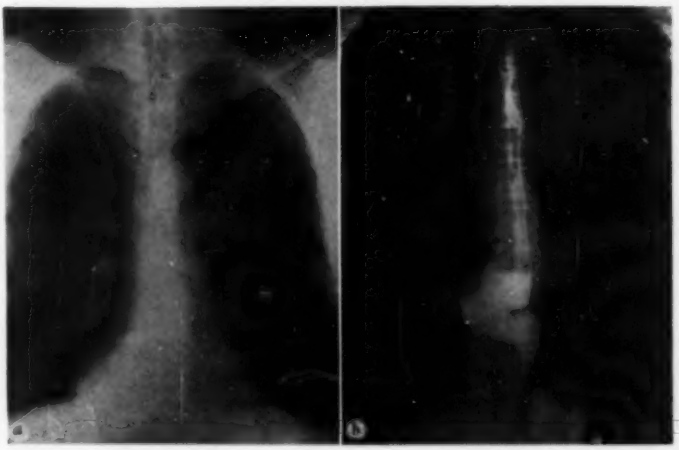

FIGURE 13a FIGURE 13b

*Figure 13a and 13b:* Leiomyoma involving two-thirds of the circumference of the lower part of the esophagus of a man 53 years old. The tumor was found at routine roentgenologic examination of the thorax; it had not caused subjective symptoms. The leiomyoma was completely removed through right transpleural posterolateral thoracotomy, and the wall of the esophagus was reconstructed.

and any extension of it into the surrounding structures. Such an operation should be carried out whether the primary growth is extrapulmonary or intrapulmonary. Some malignant lesions, such as bronchial adenomas, can be treated by lobectomy rather than pneumonectomy, when their situation in the lung permits, because they do not commonly metastasize. In some instances of peripheral carcinoma of the lung, when the lesion is small and there is no evidence of metastasis, the lesion similarly can be treated by lobectomy, particularly if the patient has limited pulmonary reserve or associated serious systemic disease or cardiac disease.

*Findings in 291 Extrapulmonary and Intrapulmonary Lesions*

In a series of 177 patients with various types of extrapulmonary intrathoracic lesions which I have treated surgically, 36 presented no subjective pulmonary symptoms. The lesions of these 36 were found by routine roentgenologic examination of the thorax during a group survey or by roentgenologic examination of the thorax during a general examination.

I shall mention only briefly the lesions of the 36 patients with circumscribed lesions found at routine roentgenologic examination and who did not present symptoms. These 36 patients presented lesions in various positions throughout the thoracic cavity. There were eight neuroblastomas, two of which were of the dumbbell

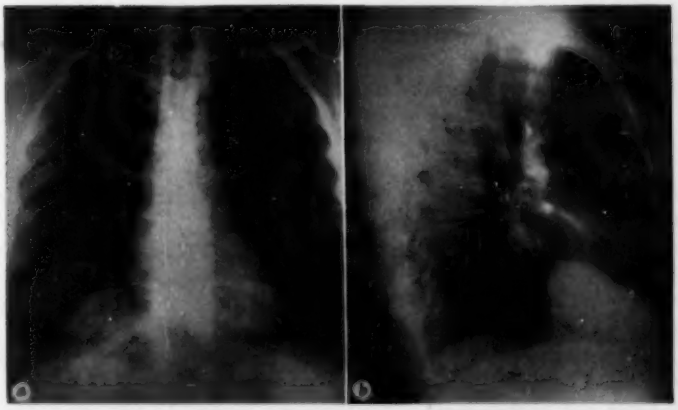

FIGURE 14a FIGURE 14b

*Figure 14a and 14b:* Diaphragmatic hernia, with herniation of a large portion of liver through the anterior dome of the right side of the diaphragm of a man 34 years old. The lesion was found by routine roentgenologic examination of the thorax; the patient had presented no subjective symptoms. The diaphragmatic hernia was repaired through a right posterolateral thoracotomy after replacement of the involved portion of liver into the abdomen.

type; one was a fibroma; five were teratomas, of which one was malignant; two were malignant thymomas; one was in intrathoracic goiter; seven were various types of cysts, most commonly coelomic cysts of the anterior mediastinum attached to the pericardium; two were granulomas of the mediastinum; one was a lipoma; two were malignant endotheliomas; one was a reduplication of the esophagus; two were leiomyomas of the esophagus; and four were diaphragmatic hernias with herniation of viscera through the right hemidiaphragm, of which two were subcostosternal (foramen of Morgagni) and contained omentum only, and two were through the right dome of the hemidiaphragm and contained part of the liver.

In addition to this series of 177 extrapulmonary lesions, 114 malignant and benign intrapulmonary lesions were discovered and removed surgically. In this special group of 114 malignant and benign intrapulmonary lesions, 16 had not caused subjective pulmonary symptoms and were found on routine examination of the thorax. Two of these were peripheral carcinomas of the lung, three were metastatic malignant lesions of the lung, one was a mixed tumor, one was a malignant bronchial adenoma, five were hamartomas of the lung and four were granulomas of the lung.

It is also of interest to note in seven patients who had inoperable primary malignant processes of the lung, proved by clinical examination and surgical exploration, the lesions were first found by routine examination of the thorax and the patients had presented no subjective pulmonary symptoms. These seven of course are not included in this series of patients who underwent surgical resection of malignant lesions of the lung.

## SUMMARY

The value of routine roentgenograms of the thorax in disclosing the presence of lesions of the thorax in patients who have no subjective symptoms suggesting intrathoracic disease is shown in the foregoing series of 291 various types of extrapulmonary and intrapulmonary lesions which I have treated surgically. In 52 of these cases the patients did not have any symptoms indicating the presence of a lesion.

The most important consideration in this group of 52 cases is that in 12 the lesion was found to be malignant. In nine of these 12 cases the tumors were early primary malignant processes, and surgical removal was achieved at a time when the results of such a procedure are most favorable. Three of the 12 malignant lesions were metastatic, and it is probable that removal will give only a palliative result.

Forty of the 52 symptomless lesions were benign, and removal

of them was attained before they had caused any serious injury to surrounding structures or had undergone malignant change.

The importance of a complete clinical examination or, if necessary, exploratory thoracotomy to establish a definite diagnosis and institution of early surgical treatment, is shown by the various types of lesions found in this series of 52 cases of symptomless thoracic tumors: 12, or 23 per cent, of the lesions were malignant.


## RESUMEN

En la presente serie de 291 tipos diversos de lesiones extrapulmonares e intrapulmonares, que he tratado quirúrgicamente, se comprueba el valor de las roentgenografías rutinarias del tórax en revelar la presencia de lesiones del tórax en pacientes que no tienen ningún síntoma subjetivo que sugiera enfermedad intratorácica. En 52 de estos casos los pacientes no tenían síntomas que indicaran la presencia de una lesión.

La lección más importante de este grupo de 52 casos es que en 12 se encontró que la lesión era maligna. En nueve de estos 12 casos los tumores eran procesos malignos primarios, y se llevó a cabo la extirpación quirúrgica en el tiempo en que los resultados de ese procedimiento son más favorables. Tres de las 12 lesiones malignas fueron metastásicas y lo probable es que la extirpación sólo dé un resultado paliativo.

Cuarenta de las 52 lesiones asintomáticas fueron benignas y se extirparon antes de que hubieran causado grave daño a los órganos circundantes o de que sufrieran alteraciones malignas.

La importancia de un examen clínico completo o, si es necesario, de una toracotomía exploratoria para establecer el diagnóstico bien definido y para iniciar un tratamiento quirúrgico temprano, fue demostrado por los varios tipos de lesiones que se encontraron en esta serie de 52 casos de tumores torácicos asintomáticos: 12, o el 23 por ciento, de las lesiones fueron malignas.


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# Discussion


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It has always been interesting and instructive to listen to a paper by Dr. Harrington and today's presentation is no exception. The evaluation of circumscribed intrathoracic lesions, particularly those causing no symptoms has become an increasingly frequent chore. As discussor, I can add nothing to the message so ably

presented by the essayist but I can, perhaps, emphasize several points of importance.

One additional diagnostic procedure should be mentioned. It is simple and has given needed information a number of times in cases both of intrapleural and extrapleural masses. It consists of anterior scalene and retroclavicular lymph node biopsy, even when these glands are not palpable. Dr. Daniels of San Francisco has described the procedure in "Diseases of the Chest," September 1949. Many of us around the Bay have used this method to good advantage.

Dr. Harrington has discussed both mediastinal and intrapulmonary tumors. In regard to the former, we should stress that there are three cogent reasons for advising early exploration: the danger of malignant degeneration; the probability of increased size with deleterious pressure on vital structures; the possibility of suppuration with extensive adherence and consequent increase in difficulty and risk of surgery.

It is often difficult to convince a patient that major operation should be undertaken for mediastinal tumors when no symptom exists. Frequently, I have demonstrated the size of the lesion to the patient and then have inquired as to his or her attitude should a mass of similar size be found in the breast, abdomen, pelvis or under the skin. This simple lesson in analogous situations often is successful.

Dr. Harrington has mentioned and I would like to emphasize the fact that benign mediastinal tumors because of position and pressure can cause nerve palsies and venous obstruction, signs ordinarily attributed to malignant growths. I removed a large left apical neurofibroma from one such young female who, pre-operatively, presented a Horner's Syndrome, paresis of the left vocal cord and left diaphragm and obstruction of the venous return with enlargement of the superficial veins over the left arm, shoulder and breast.

The tragedy of inoperable carcinoma of the lung is too well known to warrant debate. One is left with but little choice except uniformly to recommend exploration in nearly all adult patients with intrapulmonary lesions, particularly if a specific infection can reasonably be ruled out. Even when a justifiable delay is counseled, the *psychology* of watchful waiting is bad. Not infrequently, it is impossible to convince the patient of the vital necessity for interval physical and roentgen examinations.

# Considerations of the Clinical and Physiologic Factors in the Treatment of Chronic Pulmonary Conditions*

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With the advent of the antibiotic drugs and chemotherapy the dangers and complications of pneumonia, abscess, and other acute pulmonary infections have been greatly minimized, permitting a longer span of life. Witness especially the phenomenon of pneumonia, relegated from the role of "Captain of the Men of Death" to the innocuous category of benign infection. While the gratifying results of modern therapy are recognized, the ultimate gains have been questioned due to the rising incidence of degenerative pulmonary diseases. Health authorities are deeply concerned over the present inadequacy for hospitalization and rehabilitation of persons with chronic pulmonary conditions.

The present paper deals with certain fundamental and clinical aspects of chronic pulmonary diseases and their management, as observed in 600 persons from 50 to 75 years of age. The group comprises anthracosilicosis, pulmonary fibrosis, emphysema, chronic bronchitis, bronchiectasis, bronchiolitis fibrosa obliterans, pulmonary arteriosclerosis, passive congestion associated with *cor pulmonale*, and inactive tuberculosis with residual pulmonary complications. The data refer specifically to more than 100 men and women in whom the pulmonary manifestations unquestionably are related to degenerative conditions of the lungs.

In proposing a regimen of treatment, certain pathologic features of chronic pulmonary diseases are mentioned.[1,2] These concern the abnormalities of bronchi, bronchioles, parenchyma and pulmonary blood vessels, in association with the gross alterations of fibrosis, emphysema, and malposition of structure. While pleural complications are recognized as factors in the cause of degenera-

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tive diseases of the lungs, they are omitted in the present paper, since the considerations relate primarily to the mechanism of intra-pulmonary disease.

The typical features of pulmonary fibrosis are the progressive substitution of fibrous tissue for normal parenchyma. The process may be localized or general in type. With the localized form, antecedent infection such as tuberculosis and bronchiectasis may be noted; loss of resiliency with contraction is striking in the advanced cases. In generalized fibrosis there are the evidences of acute bronchitis, superimposed infection, emphysema, occlusion of blood vessels, and passive congestion. In aging persons, emphysema is a companion disease of pulmonary fibrosis.

Emphysema is a distended condition of the lungs, in which stretching of the alveoli with constriction and obliteration of the capillaries is a feature. The contributing influences relate to the loss of elasticity of the lungs due to fibrosis, and the presence of thick, tenacious mucus in the smaller bronchioles. While the pathology indicates that the fibrosis is the predominant factor, it appears that degeneration is prominent in its own right, as suggested in the atrophy of the membrane and blood vessels of the air sacs. Focal emphysema in which accumulations of carbon are found may be due to blockage of the bronchioles and alveoli.[3]

Chronic tracheobronchitis, a frequent accompaniment of pulmonary fibrosis and emphysema, is a long-standing inflammatory disease of the bronchial tree with atrophy, fibrosis, and ulceration involving the mucous membrane, cilia, and deeper structures. Bronchiectasis is characterized as a localized accentuation of chronic bronchitis, with dilatation, inflammation, and destruction of the involved mucous membrane. Chronic tracheobronchitis and bronchiectasis, occurring as parallel developments, may be a part and parcel of pulmonary fibrosis and emphysema.

Bronchiolitis fibrosa obliterans is essentially a disease of the bronchioles, in which scar tissue formation and obliteration of the lumen are characteristic. The walls of the bronchioles, in early cases, show desquamation of the epithelium and sometimes necrosis; with the advanced disease, scar tissue formation and occlusion of the bronchioles are noted. While involvement of the bronchioles is primarily a local disease, varying degrees of degeneration are usually noted in the larger bronchi and parenchyma of the lungs. The condition is important therapeutically, since with timely and adequate treatment certain bronchioles may be spared from irreparable degeneration.

Pulmonary abscess is a localized, suppurative process, with or without cavitation. The following types are noted: a) single, unilateral or multilocular; central or peripheral; closed or open with

relation to the bronchus and pleura; and b) multiple, in one or both lungs.[2] In certain cases the first alterations simulate pneumonia or pneumonitis; in others the onset is characterized as necrosis. The lesions vary in size from a pinhead to an orange; obstructive phenomena of the related bronchus and foul odor of the sputum are characteristic. A significant feature of the healing process is the tendency to localized fibrosis and bronchiolar blockage.

Pulmonary arteriosclerosis is classified as primary or secondary in type. The primary form is not clearly defined; undoubtedly there is an association with generalized arteriosclerosis. With the secondary type, as observed in emphysema, fibrosis, and long-standing passive congestion, there is the significant evidence of vascular degeneration affecting pulmonary contractility. The vascular alterations include thickening of the intima and degeneration of the media of the pulmonary artery, with partial or complete obliteration of the branches. The arterioles and capillaries may be involved, especially in the secondary form.

Passive congestion occurs with failure of the pulmonary circulation, the usual cause being mitral and aortic insufficiency with myocardial disease. The lungs, in long-standing cases, are brown in color with loss of contractility; associated pulmonary fibrosis, either as a primary or secondary condition, is not uncommon. The pulmonary capillaries are engorged, distended, and projected as loops into the alveolar spaces; certain alveoli contain degenerated epithelial cells, red blood corpuscles, and so-called heart failure cells.

It is evident from the pathology of chronic pulmonary conditions that the processes amendable to treatment are primarily bronchial, bronchiolar (mechanical and generalized), and infectious (localized) in type. The symptoms of chronic pulmonary diseases reflect, to some extent, the nature of the pathology and the associated physiologic disturbances. Cough, dyspnea, and expectoration are significant manifestations.

Cough is characterized as a phenomenon of forceful expiration, in which the medulla oblongata, the glottis, and reflexes of the bronchi participate; collections of mucus, muco-pus and foreign material are the chief precipitating influences. While the cough mechanism is recognized as an important means for the drainage and expulsion of bronchial secretions, the traumatizing influences on sensitive, diseased pulmonary tissue cannot be underestimated. In the presence of bronchospasm with impaired alveolar ventilation, the effects are accentuated because of the increased intrabronchial pressure occurring with each paroxysm. The tone, frequency, and precipitating influences in cough may be important

leads for determining the underlying disease. A "grunting" type of cough, induced during physical effort and occurring independently of infection, suggests respiratory embarrassment. Cough, as observed in the asthmatic states, is wheezy in type and indicates bronchospasm. In suppurative disease, the cough is "loose" in type, periodic, and coincident with the accumulations of sputum in the bronchi or lung. The type of expectorated material varies with the disturbances of the parenchyma and bronchial mucous membrane; as for example, glary, mucoid expectoration is characteristic of tracheobronchitis, allergic states, and uncomplicated emphysema; marked expectoration is typical of bronchiectasis and abscess and characteristically fetid in odor when associated with blockage of the bronchus. In the general consideration of cough, it would appear that infection *per se* may be an incidental factor, as compared with the primary influences of bronchospasm, emphysema and fibrosis. Dyspnea results from compensatory hyperventilation, and in association with chronic pulmonary disease suggests an element of emphysema; accentuation of the dyspnea during episodes of chest colds and fever signifies bronchospasm and decreased bronchial drainage, with or without superimposed infection.

The physical signs, as with symptoms, reflect the nature of the pathology and physiological function of the lungs. In pulmonary fibrosis, the location, degree, extension and behavior of the processes are revealed significantly. This is illustrated in cases with retraction of the affected side of the chest, narrowing of the intercostal spaces, drawing down of the shoulder, atrophy of the overlying muscles, impairment of the percussion note, exaggeration of the breath sounds, and localized scattering of medium rales; in long-standing cases, hyperresonance of the percussion note may be present over the opposite lung. Displacement of the heart and mediastinum to the affected side of the thorax is noted in advanced fibrosis. With involvement of both lungs there is the additional phenomena of generalized limitation in expansion of the chest.[1]

Pulmonary emphysema is characterized in about 50 per cent of the cases as a disease with barrel-shaped chest; widening of the intercostal spaces; an obtuse angle of the ribs; elevation of the thorax, rising as a whole during inspiration; a preponderance of abdominal type of breathing; and clubbing of the fingers in cases with marked arterial oxygen unsaturation. In the remaining group, the thorax is elongated and flattened and the respiratory excursions occur chiefly in the vertical plane, the abdominal movements being prominent during the respiratory cycle. With asthmatic states the expiratory breath sounds are somewhat prolonged and

harsh in quality; musical rales and wheezing sounds are numerous, especially in forceful breathing; indrawing of the interspaces is not uncommon. With pronounced fibrosis the inspiratory phase may be relatively exaggerated, the rales scattered, and the breath sounds distant.

The physical signs of chronic tracheobronchitis are usually associated with other processes of the bronchi or lungs; in some instances they are entirely masked. With exacerbations due to intercurrent infections, wheezing, sonorous rales and limited expansion of the chest are not uncommon. The signs of bronchiectasis include restricted movements of the affected area of the chest, with medium, coarse rales and exaggerated breath sounds of an extremely varied and changeable nature. There often is a direct relationship of the signs to the freedom and the amount of expectoration; as for example, with adequate expectoration the rales and breath sounds are prominent, in contrast with distant breath sounds and limited rales that occur during periods of blocked drainage. These typical variations of bronchiectasis are illustrated with examinations performed before and after morning expectoration. In bronchiolitis fibrosa obliterans the physical signs are rarely diagnostic. The presence of persistent fine and medium rales scattered throughout the lungs in the presence of marked dyspnea and cyanosis is suggestive; slight impairment of the percussion note occurs in certain cases. The condition may be suspected when dyspnea is out of proportion to the existing emphysema and fibrosis, and if there is no evidence of heart disease. The suggestive signs of pulmonary arteriosclerosis include increased venous pressure and accentuation of the second pulmonic sound, with distension of the right side of the heart and superficial veins. The electrocardiogram shows right axis deviation of the QRS complexes.[4] Cyanosis and dyspnea may be marked and greatly accentuated during exercise; vertigo, hemoptysis and precordial pain are not uncommon. As with bronchiolitis fibrosa obliterans, the clinical diagnosis is presumptive.

X-ray films for many years have held undisputed claim in the diagnosis of chronic pulmonary diseases, their value relating especially to the features of superimposed and static processes. Areas of increased density are characteristic of infection, atelectasis, and fibrosis; in emphysema, increased illumination of the lung fields with lowered and flattened diaphragm is noted. In general, the persistence of abnormal shadows is an indication of static disease. While the forte of the x-rays is the diagnosis of chronic structural disease and exacerbations of acute processes, there is increasing doubt as to their infallibility in determining the presence of disability. Interestingly, the status of the x-ray film in

certain cases is in parallel with the value and limitations of the medical history and physical examination; as for example, while the x-ray and the physical signs indicate the presence of disease, the finer aspects of regression or extension may not be fully emphasized. Hence, need for physiological testing has become evident.[4-7]

The fundamental mechanism of disturbed pulmonary function is interference with the passage of air in the lungs and retardation in the drainage of secretions. The problem is elucidated by the classification of emphysema based on the physiological relationship of the volume of residual air to total lung volume, and from ventilation measurements that indicate the degree of impairment due to the mixing and dilution factors; namely, vital capacity and residual air.[4] In pulmonary fibrosis, occurring in persons of various age groups, all degrees of emphysema are noted, and it is interesting that no apparent correlation exists between the roentgenographic stage of the disease and the associated emphysema. It appears significant that in about 30 per cent of the cases of fibrosis due to silicosis, emphysema is not an important cause of faulty pulmonary function. However, in the presence of an upper respiratory tract infection causing gravitation of secretions into the dependent bronchioles, especially when associated with bronchospasm, there is an increased volume of residual air. This constitutes a definite handicap in breathing, as the degree of compensatory hyperventilation is reduced and the patient becomes incapacitated during the attack.

Vital capacity and maximal breathing capacity are important ventilation measurements. Vital capacity decreases as the degree of emphysema increases. Wide individual variations limit the value of the measurement in certain cases; as for example, the lowered vital capacity of 2,000 cc. to 3,000 cc. may exist with no manifest emphysema (determined by quantitative measurements of residual air), as well as in cases of moderate or even advanced emphysema.[4] Maximal breathing capacity is related to the degree of emphysema in a manner similar to the correlation between vital capacity and emphysema. If the maximal breathing capacity* is less than 40 liters per minute emphysema of a significant amount is present (residual air 35 per cent or more of total lung volume), and if the maximal breathing capacity is over 100 liters per minute the impairment, in most cases, is not due to emphysema. The following physiologic aspects are especially significant in treatment: The residual air per cent of total lung volume increases principally

*This measurement requires the use of special apparatus permitting a minimum degree of breathing resistance; a standard basal metabolism machine is unsatisfactory.[4]

at the expense of the complemental air per cent of total lung volume;[6,7] and the percentage portions of the lungs occupied by supplemental air (reserve) and tidal air remain nearly constant, regardless of the degree of emphysema. Accordingly, with far advanced pulmonary emphysema the breathing reserve may be markedly reduced, since the depth of inspiration is increased only slightly above that of the resting state.

In addition to the physiologic problems directly related to the emphysema *per se*, there is faulty air distribution in the lungs. With this type of pulmonary dysfunction, impairment in breathing results from the unequal aeration and perfusion caused by the fibrotic and obstructive changes in the lungs. Poor distribution of air lowers the arterial $pO_2$ and per cent oxygen saturation due to the large drop in mean $pO_2$ between the alveoli and the arterial blood. In some cases the increased mean gradient of pressure for oxygen between the alveoli and arterial blood approaches 50 mm. Hg. (normal, 5 mm. Hg.), and the average resting arterial oxygen saturation is moderately reduced but poorly correlated with pulmonary function impairment. It is interesting with respect to treatment that the arterial oxygen values obtained during mild exercise may reveal significant differences with respect to disability and the degree of emphysema, as demonstrated when the arterial oxygen value is compared with the resting level. Comparisons between step-up and treadmill exercise in 54 patients, using arterial blood and respiratory gas exchange measurements, provided important information relating to the response to physical effort[8] (Table I). In 17 cases, the treadmill exercise produced a slight decrease in arterial oxygen saturation; in 24, there was a slight increase; and in 13 cases, there was a significant increase of oxygen saturation (3 per cent or more).

The one minute step-up test is a more severe form of exercise for some patients with an advanced degree of pulmonary function impairment than the treadmill, since, with the latter, measurements are taken after 10 minutes, and the subject becomes ex-

TABLE I

Measurements of Respiratory Gas Exchange During Rest and Exercise in 54 Cases of Pulmonary Fibrosis and Emphysema.

| | *Art. $O_2$ Sat.* | *$O_2$ Cons.* | *$CO_2$ Output* | *Ventil. Vol.* |
|---|---|---|---|---|
| | Per Cent | cc/sq. m/min. | | l/sq. m/min. |
| Rest | 92.9 | 145 | 118 | 4.7 |
| Step-up | 91.6 | 474 | 355 | 12.2 |
| Treadmill | 93.4 | 484 | 400 | 12.6 |

hausted if the treadmill is set too fast or with too much tilt. In 36 of the cases with a significant degree of emphysema (residual air over 35 per cent of total lung volume), the arterial oxygen saturation was decreased more definitely with the step-up exercise,[8] although the oxygen uptake and carbon dioxide output were increased about the same with the two forms of exercise (Table I). The data also indicate that the pulse and respiratory rates, the blood pressure, and percentage of oxygen removed from the air breathed do not vary significantly between the two methods of exercise. It was striking in the physiologic studies of treadmill exercise that in about 70 per cent of the cases the respiratory gas exchange was improved, as compared with the figures obtained during bed rest.

The present treatment of chronic bronchitis, emphysema, pulmonary fibrosis, anthracosilicosis, bronchiectasis, bronchiolitis fibrosa obliterans, and pulmonary arteriosclerosis is based on the accepted measures of prevention and the newer concepts of physiologic rehabilitation, as follows: 1) symptomatic relief; 2) measures that favor elimination of infection and abnormal secretions from the bronchial passages; and 3) regulation of the type of respiration and general physical activities of the body. The effective application of procedures requires a critical evaluation of clinical and physiological data.

Medical direction should include the correction of metabolic states, especially obesity, since excessive weight is a burden on the failing pulmonary circulation. It is desirable also to eliminate chronic infections of the teeth and respiratory passages to avoid the gravitation of pus and abnormal secretions into the dependent parts of the lungs. Sinus disease and allergic states may accentuate the development of bronchial disease and emphysema, and with their first appearance it becomes essential to avoid sensitivity reactions, dust hazard, dampness, and extremes in temperature. Heart disease and hypertension are predisposing to pulmonary disability[9] and appropriate treatment is indicated.

Therapeutic rest in bed requires careful consideration; its value is unquestioned but regulation is required. Rest is desirable, within limitations, for superimposed infections, including tuberculosis, but definitely is not indicated in uncomplicated emphysema, fibrosis, bronchitis and bronchiectasis. It should be emphasized that restriction of physical activities may be contraindicated, since the control of respiratory movements favors disuse of the lungs, resulting in impaired physiological function. In terms of every-day living, excessive use of the rocking chair becomes as detrimental as physical exertion is harmful in active pulmonary tuberculosis. Indeed, the retarded movement of air in the lungs

with the lessened discharge of bronchial secretions may be the penalty of unnecessary physical rest. Contrarily, graduated or regulated exercise expedites the resolution of intercurrent pulmonary infections and facilitates normal body functions, and it should be recommended with the first lessening of the acute episodes. Gymnastics, with deep breathing exercises, employed on rising from bed in the morning, are sometimes valuable, certainly in favoring adequate pulmonary ventilation; holding the abdomen from below upwards during a series of forceful respirations and the use of controlled cough practiced at the end of full expiration may be extremely helpful.[10] Fatigue and chest discomfort with increased pulse and respiratory rates, occurring with exercise, call for limitation of activities; stair climbing and walking rapidly, especially against the wind, should be avoided. Exercise should be curtailed or stopped with the first evidence of circulatory embarrassment.

The value of drugs alone in chronic pulmonary diseases is disappointing, and often the side effects are prominent. The expectorants, including creosote and the iodides, are disturbing to the gastro-intestinal system and there is some question as to their secretion in the impaired bronchial mucosa. Sedative drugs such as morphine, codeine and dilaudid retard bronchial drainage and usually are not valuable except for the control of hemoptysis, racking cough and thoracic pain. Aminophyllin may be useful in cases of bronchospasm and pulmonary hypertension. In failure of the circulation due to primary disease of the myocardium, digitalis, theobromine derivatives and mercuhydrin are valuable, especially with oxygen therapy. Penicillin, aureomycin and terranycin are effective in recurrent non-tuberculous infections (see section on intermittent positive pressure breathing treatments).

Emphasizing the limitations of drug therapy in chronic pulmonary conditions is the observation that two more or less distinct types of disease are present: a) processes that are chiefly static and due primarily to fibrosis and so-called pathologic emphysema; and b) those that are somewhat reversible in type, caused by intercurrent infections, blockage of the bronchioles, and collapse of the alveoli. With the hypothesis that certain manifestations are due, in part, to reversible processes which exert both mechanical and physiologic influences, the use of dynamic therapy in the form of respiratory exercise has been employed. The method consists of intermittent positive pressure breathing with oxygen (100 per cent) and the supplementary use of aerosols, consisting of bronchodilator drugs, a wetting agent, antibiotics, and diaphragmatic elevation.

Interest in the use of intermittent positive pressure breathing

treatments (IPPB) was aroused by the studies of carbon monoxide poisoning, acute pulmonary edema, barbiturates poisoning,[11] anthracosilicosis,[12,13] and more recently poliomyelitis.[14] It has been observed that IPPB facilitates alveolar aeration by: 1) decreasing the mean oxygen transfer gradient (alveolus to arterial blood); 2) increasing arterial $pO_2$; and 3) favoring a rise in the arterial oxygen saturation. Applying the method in chronic pulmonary diseases, bronchodilator drugs are used for their supplementary action, to facilitate the passage of air through the bronchioles and to encourage the drainage of secretions; a wetting agent is employed, in selected cases, with the possibility of dislodging thick, tenacious secretions; and the antibiotic drugs are used to control persistent infection.

The regimen of IPPB is as follows: The apparatus consists of a cycling valve (either pressure sensitive* or flow sensitive**), mouthpiece or mask, nebulizer to provide intermittent positive pressure, and other units for introducing oxygen, 100 per cent, and aerosol medication into the respiratory passages. The bronchodilator drugs, isuprel (0.05 per cent solution) and vaponefrin (2.25 per cent solution), are administered singly in doses of 8 drops diluted in 8 drops of normal saline. The wetting agent, ceepryn, non-ionic type, is given in doses of 3 drops of a 1-1000 solution added to the bronchodilator drug and normal saline solution. The IPPB treatments, with aerosols, are administered two or three times daily, for periods of 15 or 20 minutes each, depending on the patient's comfort and reaction; with fatigue or palpitation the periods of treatment and the dosage of the bronchodilator drugs are reduced. The regimen is continued for two to three weeks, or longer, depending on the results. With marked pulmonary expectoration and bouts of fever uncontrolled by aerosol therapy, 200,000 units of penicillin are administered parenterally two times daily; and with no improvement, aureomycin, 200 mg., is given orally every four hours. In tuberculous cases, streptomycin, 1.0 gm., is administered parenterally, once daily. The antibiotics are rarely used by aerosol.

Diaphragmatic elevation is induced by abdominal compression.[15,16] With this mechanism there is displacement of the diaphragm to the higher and more effective range of expiration.† Useful as an adjunct for pressure breathing, it is especially indicated for the long, flat type of chest with a preponderance of

*The Burns valve (pneophore) is manufactured by the Mine Safety Appliances Company, Pittsburgh, Pa., and **the Bennett valve is made by J. J. Monaghan Company, Denver, Colorado.

†The apparatus (diaphragm lift) is manufactured by Nulty-Coggins, 1921 Chestnut Street, and C. J. Pilling and Son Company, 3451 Walnut Street, Philadelphia, Pennsylvania.

vertical respirations, racking cough and difficult expectoration. The primary purpose of diaphragmatic elevation is to facilitate bronchial drainage, favor the movement of air in and out of the lungs, and to decrease the traumatizing effects of cough.

The results of IPPB, observed in the symptomatic treatment of chronic pulmonary diseases, have been definite in about 60 per cent of the patients affected with dyspnea, hard racking cough, and tenacious sputum. Improvement consists of easier breathing at rest and during exercise, with lessened cough and expectoration; a remarkable feature, in certain cases, has been the expectoration of large quantities of pus before striking improvement of breathing is noted. Certain patients, following a period of treatment, have been able to walk on the level with no manifest dyspnea, sometimes up grades varying between 3 and 10 per cent. Improvement has been most striking in the cases with a moderate admixture of emphysema and fibrosis, with or without chronic bronchitis. The failures have occurred chiefly in weakened patients with advanced disease.

While the relief from dyspnea, cough and expectoration is an index of improvement, it is realized that psychogenic factors must be excluded. Accordingly, the various observations have been evaluated as follows: In the physical examinations the appearance or disappearance of rales with improved diaphragmatic and thoracic movements has been noted, especially in emphysema associated with bronchitis. The most remarkable alterations have related to the disappearance of musical rales in the asthmatic states of bronchitis and emphysema, and the appearance and subsequent disappearance of rales as recovery occurred in congestive cases. These phenomena evidently are related to the evacuation of bronchiolar secretions, lessened bronchospasm, and improved alveolar aeration. In the roentgenograms and fluoroscopically the extent or type of structural disease was not modified significantly, but increased illumination of the lung fields in congested areas was common.[9] It was interesting that improved illumination often occurred in parallel with the modification of physical signs.

In the physiologic studies following a course of IPPB treatments the maximal breathing capacity showed an average increase of approximately 20 per cent; in certain cases an increase of more than 100 per cent was noted. Interesting data are tabulated in Table II: The average figures on the determinations of vital capacity and maximal breathing capacity (100 cases) are recorded in two groups: 28 cases with an insignificant degree of physiologic impairment, and 72 cases with a significant degree of emphysema. There was a definite increase in the maximal breathing capacity

TABLE II

Measurements in 100 Cases of Silicosis Before and After One IPPB Treatment

| NO. OF CASES | 28 | 72 |
|---|---|---|
| *Degree of Emphysema* | *Insignificant* | *Significant* |
| VITAL CAPACITY CC. | | |
| Before | 4137 | 2801 |
| After | 4202 | 3360 |
| Change | +1.6% | +20% |
| MAXIMAL BREATHING CAPACITY - L./MIN. | | |
| Before | 105.5 | 47.2 |
| After | 120.6 | 61.2 |
| Change | +15.1% | +29.8% |

of both groups (15.1 per cent and 29.8 per cent, respectively). It appears in the group with emphysema that the increase in maximal breathing capacity, after IPPB, was greater than in the non-emphysematous group, and correspondingly, greater symptomatic improvement was noted. While the vital capacity was increased significantly in the group with emphysema (20 per cent), there was no striking change in the group with no emphysema (+1.6 per cent).

The method of measuring maximal breathing capacity and vital capacity before and after treatment in two cases is illustrated by Figure 1. In each instance there was marked improvement in the maximal breathing capacity after one combined IPPB treatment with vaponefrin, considered an indication of the degree of bronchospasm present and likewise an indication of the response that may be expected from treatment of two or three weeks' duration. It has been observed even in cases without emphysema (Fig. 1, case 271) that the vital capacity is increased as the bronchospasm is relieved, and in almost every case the MBC is improved, often in a marked degree as shown in Figure 1. With reference to the mechanism of breathing, it should be noted that subjects must breathe rapidly in order to record a normal MBC value, the approximate rate being 80 times per minute; some individuals are unable to breathe rapidly, as shown with the tracing of case 280, Figure 1, where 40 per minute was the maximal rate obtained. It is significant in this case that the rate increased to 65 per minute with a large increase in the MBC following one treatment with IPPB.

The physiologic measurements indicate that the decreased breathing resistance, especially on inspiration after IPPB treat-

ment, is primarily responsible for the improvement of subjective dyspnea. It has been evident in the studies that IPPB does not influence the oxygen transfer in the normally ventilated alveolus, and there is no effect on the alveolus, still perfused but unventilated, as in complete blockage of the bronchioli. Furthermore, there is no improvement in the function of alveoli when the ventilatory capacity is preserved but perfusion is lacking. It appears that the application of positive pressure breathing (IPPB) during inspiration compensates for the limited ventilation of emphysema. Accordingly, with the improved aeration the per cent of effective tidal air is reduced by increased ventilation of the physiological dead space, including the alveoli ventilated but not perfused; however, the volume of effective tidal air is increased due to the mechanism of hyperventilation. The combined influences of the bronchodilator drugs, the antibiotics and the wetting agent accentuate the effects of IPPB by direct action on the bronchioles and their contents.

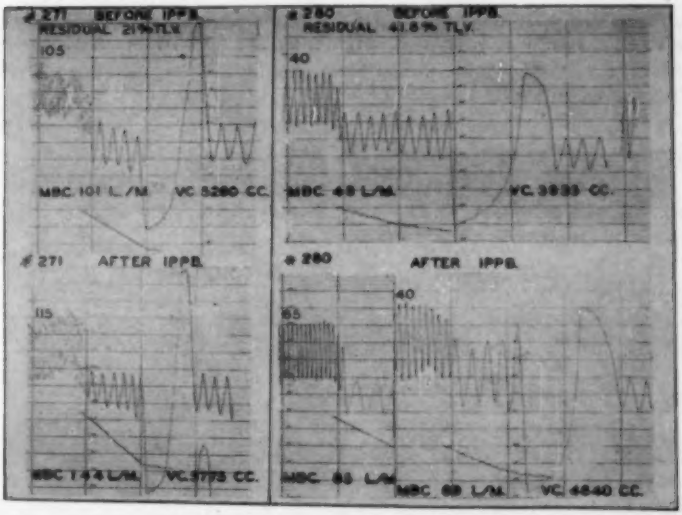


FIGURE 1: Spirograms of vital capacity and maximal breathing capacity from an emphysematous (case 280) and non-emphysematous patient (case 271) before and after one treatment with intermittent positive pressure breathing combined with vaponefrin by nebulization. Note the shorter time required for blowing out supplemental (reserve) air after treatment in each case. The increase noted in maximal breathing capacity is a measure of the degree of bronchospasm present. The spirogram tracings were recorded on a suitable type of mechanical apparatus for measuring maximal breathing capacity, as the resistance encountered with rapid deep breathing was minimal due to the employment of a high velocity valve, a large bore tubing and a large spirometer bell.[16] Time intervals equals 12 seconds between vertical lines.

In the present consideration of therapy the static, as well as the reversible nature of chronic pulmonary conditions has been stressed. The pathology and the clinical features indicate that obstructive phenomena of the bronchioles and air sacs participate closely in the development of symptoms. It appears that intermittent positive pressure breathing treatments with the supplemenary use of bronchodilator drugs provide satisfactory means for pulmonary rehabilitation, accomplished chiefly through the action of improved bronchiolar drainage and alveolar aeration. Evidently, the symptomatic relief and physiologic improvement are not related to a reduction in the type or extent of the structural disease.

## SUMMARY

1) The pathology and clinical features of chronic pulmonary disease are reviewed with special reference to the hypothesis that certain manifestations and disturbances of function are related to reversible disease.

2) The mechanism of intermittent positive pressure breathing (IPPB) treatment with the supplementary use of bronchodilator drugs, a wetting agent, antibiotic drugs and diaphragmatic elevation is discussed.

3) Subjective improvement is manifested by lessened cough, dyspnea and expectoration.

4) Objective improvement is determined with physiologic methods utilized before and following a regimen of intermittent positive pressure breathing treatments.

5) The studies indicate that improved drainage of the bronchioles with the active movement of air in and out of the alveoli, as accomplished with IPPB, provides a dynamic and effective means for the treatment of chronic pulmonary conditions.

## RESUMEN

1) Se repasan los rasgos patológicos y clínicos de enfermedades pulmonares crónicas con especial referencia a la hipótesis de que ciertas manifestaciones y desarreglos funcionales están relacionados con enfermedades reversibles.

2) Se discute el mecanismo de la terapia de respiración con presión positiva intermitente (RPPI) con el empleo suplementario de drogas broncodilatadoras, agentes humectantes, drogas antibióticas y elevación del diafragma.

3) Se manifiesta la mejoría subjetiva por la disminución de la tos, la disnea y la expectoración.

4) Se determina la mejoría objetiva mediante métodos fisioló-

gicos empleados antes y después del régimen de tratamientos de respiración con presión positiva intermitente.

5) Estos estudios indican que la mejoría en la canalización de los bronquiolos debida al movimiento activo del aire que entra y sale de los alvéolos, que se obtiene con la RPPI, provee un medio dinámico y eficaz para el tratamiento de estados pulmonares crónicos.

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# Discussion

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This paper is of great interest particularly when taken in conjunction with the remarks of Doctors Barach, Levine and Westerman last Friday, June 23. For the past ten years I have been concerned by the fact that most of the advances in chest diseases seem to have been brought about by the thoracic surgeons whose progress since the development of intra-tracheal anaesthesia and the use of antibiotics has been phenomenal. It is refreshing, therefore, to hear an address such as that given by Doctor Gordon, who has stressed the fact that chronic non-tuberculous chest diseases have a hopeful aspect quite aside from the surgical one. The more accurate appreciation of the physiology and the pathology of these chronic diseases, together with the use of antibiotics, has made the picture much more favorable.

Quite rightly, it seems to me, Doctor Gordon stresses the importance of physical signs as opposed to x-ray evidence. The physical signs of emphysema are very striking and it is likely that the skilled practitioner can determine much more with his fingers and with his stethoscope about the true function of the underlying lung than he can from looking at an x-ray film, valuable though the latter may be. I suspect that too few clinicians appreciate the extreme importance of repeated study of the sputum and its gross appearance in the diagnosis of bronchiectasis. Similarly, too few take the trouble to examine bronchiectatic patients while they are inverted and taking their postural drainage, when by simple palpation the diseased area can be easily localized, often quite as well, I think, as with lipiodol. Certainly this method of localization has an enormous advantage in not confusing future evaluation when it may be important to localize the disease accurately for surgical purposes.

After all, as Doctor Gordon says, there are only four fundamental principles of importance in these chronic non-tuberculous chest diseases. One: the unobstructed airway. After bronchoscopy has ruled out such possibilities as tumors, strictures and the like, it is reasonable to assume, especially in conjunction with careful physical examination, that the obstruction exists in the finer bronchi and bronchioles. Here the use of epinephrine in one to two-hundred to three-hundred dilution by aerosol suffices to open the airway temporarily and can be employed every two or three hours if necessary for this purpose.

The second principle is adequate drainage. After making sure that the airway is unobstructed, complete inversion of the patient

every two or three hours during the day over a period of many months produces striking amelioration of the symptoms. An excellent method is to have the patient kneel on a chair, hands on the floor, and bend his elbows until his chest is completely inverted. This of course applies to basal lesions.

Having cleared the airway and obtained adequate drainage, a great deal of infection will take care of itself. I would reserve the use of antibiotics for acute episodes in the course of these chronic non-tuberculous pulmonary diseases. Their results in producing bacterial resistance is undesirable since it leaves the patient without recourse during periods of acute infection. Whether the bacterial flora is more sensitive to penicillin or streptomycin can be determined by the gram stain. Having cleared the airway, provided adequate drainage and made certain that infection has been controlled, one is ready to begin the re-establishment of the normal mechanics of respiration. In these patients the diaphragm is often low and immobile. Since diaphragmatic function contributes 60 per cent to vital capacity under normal conditions, the motion of the diaphragm must be restored. Sometimes this can be done by a belt, as Doctor Gordon suggests. At other times it is better to have the patient lie on his or her back and consciously lift the abdominal wall, at the same time refraining from costal breathing until diaphragmatic respiration is re-established. The methods of accomplishing this, of course, are many and the results can easily be checked by fluoroscopic observation.

Certainly the most important factor in the care of these patients is long continued observation similar to that employed in the care of tuberculous patients. It should extend over a period of years. These patients tend to relapse into their old bad habits, they tend to neglect themselves and they forget what is told them. Finally, the treatment for these chronic patients should be simple, so they can use it themselves. Only this way will they be able to achieve the maximum ability to care for themselves and learn to live with their diseases, under the most favorable circumstances.

# Congenital Chondrosternal Depression (Funnel Chest) Its Treatment by Phrenosternolysis and Chondrosternoplasty

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The deformities of the anterior chest wall, commonly referred to as funnel chest, pigeon chest and Harrison's grooves have been described in the literature since the sixteenth century. Surgical attempts to correct the depression of a funnel chest began at the turn of the last century. In 1939 Ochsner and DeBakey[6] published an excellent review of the various operative procedures that have been described for the correction of this deformity together with a report of a personal case. About the same time Brown[3] described two different surgical procedures for the treatment of "pectus excavatum" in young infants and in older children. Since then Lester,[4] Sweet[8] and Ravitch[7] following the methods described by Brown have reported satisfactory results.

It is unfortunate that a large variety of names, based entirely on the outward appearance of the anterior chest wall, exists for each deformity. The author's nomenclature is based on the anatomical and etiological factors. Deformities of the anterior chest wall may be congenital or acquired as from trauma or disease. This publication will not consider those deformities of the anterior chest wall of the acquired type or those which result from failure of sternal fusion or malformation of the sternum or ribs. Only those deformities which are considered by the author to result from a congenitally abnormal development and function of the diaphragm will be discussed. The nomenclature proposed and followed by the author is as follows:

1) Congenital chondrosternal depression (funnel chest).
2) Congenital chondrosternal prominence (pigeon chest).
3) Congenital chondrocostal grooves (Harrison's grooves).

Each type is further modified by the term mild, moderate and severe depending upon the extent of the deformity.

Numerous theories, unsupported and questioned, have been advanced to explain these deformities of the anterior chest wall. A few of the more frequently mentioned causes described in the literature are rickets, obstructive respiratory conditions and abnormal intrauterine pressure. The author proposed in a previous publication[2] that anterior chest wall deformities are produced by

the abnormal pull of a congenital abnormally developed diaphragm on its anterior chondrosternal attachment. The normal diaphragm consists of four major muscular divisions which are embryologically derived from the anterior septum transversum, the two lateral pleuroperitoneal membranes and the dorsal mesentery (see Figs. 1 and 2). These four muscular segments of the diaphragm, each separated by a raphe, arise at the lower bony circumference of the thorax and are inserted into the central trefoil tendinous portion. Like all muscles, the contraction of this circumferential musculature with its radiating fibers exerts a pull on its insertion, namely, the central trefoil tendinous portion of the diaphragm. This contraction causes the diaphragm to descend without producing any pull on any part of the thoracic cage. The contractions of a normally developed diaphragm are responsible for the normal changes in the development of the thorax which convert the infantile round chest into its elliptical shape during the first year of life.

Thoraces with deformities of the chondrosternal area have been found to be associated with abnormally developed diaphragms, particularly of the anterior portion which is developed from the septum transversum.[2] The diaphragm associated with the chondrosternal depression differs from the normal diaphragm in that the anterior portion is deficient and tendinous, with a paucity of muscular fibers. In infants the contraction of such a diaphragm during inspiration produces a retraction of the anterior chest wall by the pull of this tendinous attachment to the gladiolar-xiphoid junction of the sternum (see Fig. 7). During expiration this inward

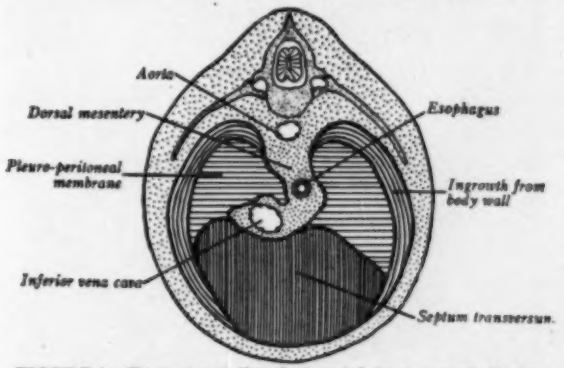


FIGURE 1: (From Arey's Developmental Anatomy). 1. Septum transversum which becomes the anterior portion. 2. Pleuroperitoneal membranes which form the lateral portions. 3. Dorsal mesentery which forms the posterior portion. 4. Muscular ingrowth from body wall which contributes to the circumferential muscular portion.

pull is released and the elastic mobile anterior chest wall recoils back to its resting position. The degree of retraction of the chondrosternal area during inspiration will vary in direct proportion to the deficiency of the anterior segment of the diaphragm. This variation will result in a mild to a very severe depression. These depressions are usually central but may be unilateral; or one side may be more severe than the other. As the infant grows older, the mobility of the retraction progressively lessens as the anterior chest wall becomes less elastic and more ossified. After the age of three, the depression of the chondrosternal area is fixed by deformed angulated cartilages and retracted sternum (see Fig. 12). The apex of the depression is at the gladiolar-xiphoid junction. In congenital chondrosternal prominence, the anterior portion of the diaphragm developed from the septum transversum was found to be fully developed but tendinous in character and contained comparatively few muscular fibers. In contrast, the lateral portions derived from the pleuroperitoneal membranes were very muscular and thick. The membranous portion instead of being trefoil assumed the shape of the letter V (Fig. 3). In infants, contraction of such a diaphragm produced a relative retraction of only the lower one-half of the chondrosternal area while the upper one-half remained round and immobile. This infantile rotundity re-

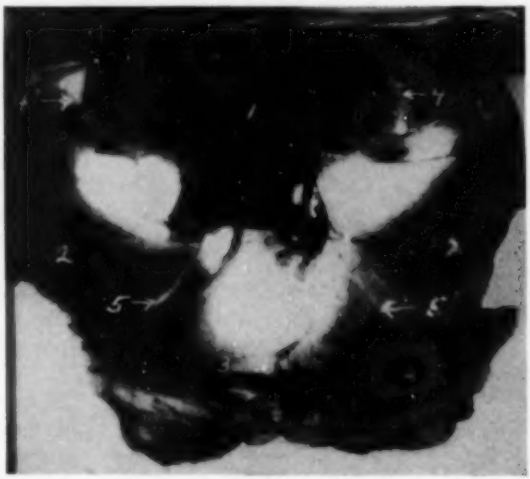


FIGURE 2: Photograph of normally developed diaphragm from a normal chest. 1. Posterior portion. 2, 2'. Lateral portions. 3. Anterior portion. 4,4'. Costovertebral trigones separating the posterior portion from the lateral portions. 5, 5'. Raphe separating the anterior portion from the lateral portions.

mains all through life as a relative prominence which is wrongly called pigeon chest. This anterior chest wall deformity is not comparable to the anterior chest wall of a fowl. The mechanism and development of this deformity was fully described in a previous publication.[2] The diaphragm associated with chondrocostal grooves (Harrison's grooves) has not been studied or observed. In infants these grooves are also synchronously produced during inspiration with contraction of the diaphragm but is observed to be fixed in older children. The author injected the phrenic nerve with 1 per cent novocaine in infants with anterior chest wall deformities to study the effect of the diaphragmatic contraction.[2] It was found that when the hemidiaphragm is paralyzed the chondrosternal area on the same side is immobile and the retraction on the other side continues during inspiration. This procedure thus far has been the strongest evidence for the advancement of the diaphragmatic theory as the cause for these anterior chest wall deformities.

These congenital defects are often not noted until the infant


FIGURE 3: Photograph of the abnormally developed diaphragm of a 14 month old infant with a congenital chondrosternal prominence. 1. Posterior portion. 2, 2'. The strong muscular lateral portions. 3. The tendinous thin anterior portion. 4, 4'. The costovertebral trigones.

is several months old although they are present at birth. Some go unnoticed during their entire life. One mother was convinced that it was unnecessary to operate on her four year old daughter for a mild chondrosternal depression only after it was pointed out to her that she herself had a more severe depression about which she was unaware. This mother had been free of symptoms all her life and never knew that she had any abnormality of her chest. Some pediatricians have stated that they have seen the deformity in early infancy which later "disappeared" as the child grew older. This is probably due to the fact that the musculature of the anterior portion of the diaphragm had not yet been fully

Fig. 3

## Phrenosternolysis

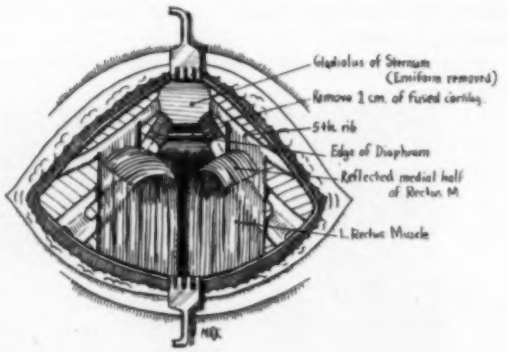


Fig. 4

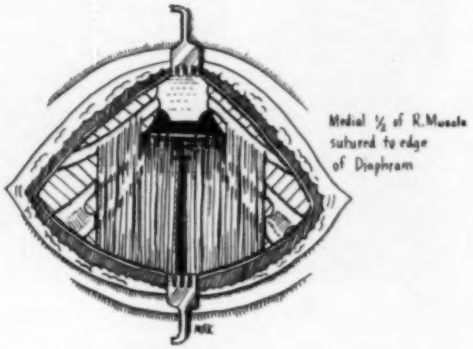


Fig. 6

developed thus producing the inspiratory retraction. As the infant grew older, this portion of the diaphragm continued to develop and its pull was transferred from the anterior chest wall to the anterior membranous portion of the diaphragm.

Infants with only congenital anterior chest wall deformities do not have cardiorespiratory disturbances in spite of the paradoxical motion of the anterior chest wall. When signs of cardiorespiratory embarrassment are present, other congenital anomalies, usually cardiac, are responsible. Therefore, the only indication for surgical treatment in young infants with a mobile inspiratory retraction of the anterior chest wall is to prevent or diminish the subsequent fixed immobile depression of the chondrosternal area which will develop later. Theoretically, the earlier the infant is treated surgically the less deformity will result. If untreated, as the infant grows older the mobility of the contraction progressively lessens and the depression becomes progressively more fixed. In children with a fixed depression, during the period of greater physical activity when greater demands are placed on the cardiorespiratory system, symptoms such as dyspnea on exertion, palpitation, precordial pain, easy fatiguability and dizziness appear. There is no relationship between the depth of the depression and the presence or severity of cardiorespiratory symptoms. Although severe deformities are consistent with normal and strenuous activity, in the author's experience patients with even moderate depressions

FIGURE 7 (Case 2): Age 5 months. Illustrates the inspiratory retraction of the chondrosternal area and epigastrium.

may show signs of interference with normal physical activity. Master and Stone[5] believe that these symptoms are "neurogenic or the result of inadequacy of the small heart in its response to emotional stress." However, when this type of patient has been treated by surgery and the depression removed, not only have the symptoms disappeared but the patient returned to strenuous activity without any further complaints. These cardiac symptoms must be probably due to the effects of abnormal rotation of the heart and great vessels or the restriction and pressure on the heart by the depressed chondrosternal area. In the adolescent, particularly males, emotional symptoms and maladjustment are often present because of the deformity. In severe cases that have not been relieved by reassurance and psychotherapy, they have been cured of their symptoms with surgical treatment.

There are usually no physical signs in infants except for the inspiratory retraction of the chondrosternal area of the chest. In children, one finds the typical posture, that is, the thin asthenic type with the head held forward, shoulders rounded and the anterior chest wall flat and depressed. Tachycardia, sinus arrhythmia, apical systolic murmurs and often an orthostatic pulse and blood pressure are the most common cardiac signs. The electrocardiographic abnormalities are low voltage, deviation of electrical axis, inverted T waves in leads three and four. Immediate post-operative electrocardiographic examination failed to

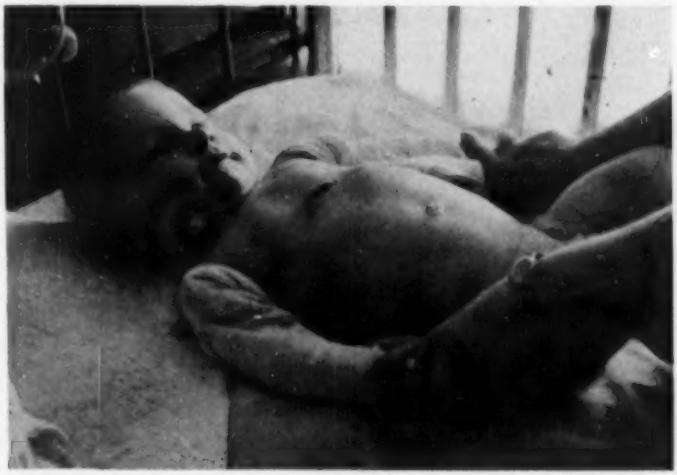

FIGURE 8 (Case 2): Five days post-operative, illustrating the incision and improvement during inspiration.

show much change although symptoms and signs were greatly relieved.

### *Treatment*

Brown in 1939 first reported a simple operation to relieve the condition in infants by bluntly separating the diaphragmatic attachment to the sternum and separating the "retrosternal ligament" from the posterior surface of the sternum. He laid particular emphasis on this step because he found this ligament greatly thickened and it appeared to him as the factor in the mechanism of this deformity. In his experience, the author has not been able to confirm these findings and, therefore, has modified Brown's procedure. After separating the diaphragm from its anterior attachment to the gladiolar-xiphoid junction and medial ends of the lowest cartilages, the freed anterior edge of the diaphragm is sutured to the freed edge of the recti muscles. In this manner the pull is transferred from the bony chest to the soft recti muscles of the epigastrium. Following the operation, the chondrosternal area shows immediate improvement in the retraction during inspiration but the soft epigastric area continues to be retracted (see Fig. 8). This operation which the author has termed phrenosternolysis is reserved for infants with moderate or severe inspiratory chondrosternal retraction. When the deformity is first seen at birth, it should be observed for several months and if no improvement is noted in the depth of the inspiratory depression, surgery is indicated as soon as possible. If it is severe it is best to operate at once. The youngest infant that was operated on in this series was five months old and the oldest was two and a half years. This procedure is advocated because it is comparatively simple and will prevent the far more extensive plastic procedure of chondrosternoplasty later. The details of the operation which is now practiced by the author in infants will be described below together with five case reports.

### *Phrenosternolysis*

A curved skin incision is made over the gladiolar-xiphoid junction of the sternum as is shown in Figure 5. The rectus fascia is incised and the medial halves of the recti muscles which are attached to the lower costal cartilages are dissected free on each side. The lowermost costal cartilages on each side are resected for about 1 cm. beginning at the gladiolar-xiphoid junction (see Fig. 4). This facilitates the exposure of the depressed xiphoid which is freed from the gladiolus. The gladiolus of the sternum is elevated with a hook. The diaphragmatic attachment is separated from the sternum to the cut edge of the lowermost car-

tilages by blunt dissection to avoid the pleural reflections. The retrosternal fascia and lower portion of the pericardium are separated from the lower posterior surface of the sternum with the aid of a small Kelly clamp. The upper edges of the medial halves of the recti are then sutured to the free edge of the diaphragm

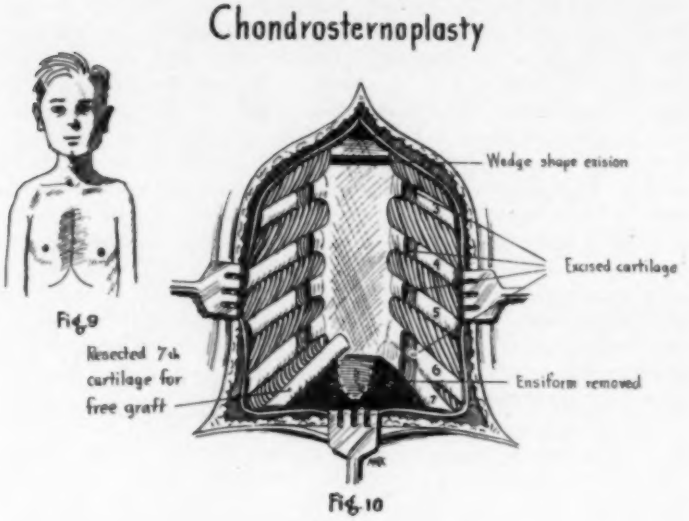


Fig. 9

Fig. 10

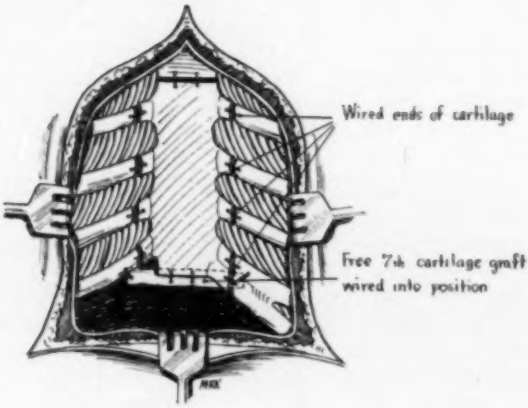


Fig. 11

by interrupted fine silk sutures as is shown in Figure 6. This is considered by the author as a very important step in the operation because it furnishes good muscular fibers to the deficient tendinous anterior portion of the diaphragm for its future growth and development. It also closes a defect which potentially may become a site for herniation. The fascia is sutured with interrupted fine silk and the skin is closed with interrupted silk. In very severe cases, a wire traction suture through the lower end of the gladiolus is brought out through the skin and is attached to a Jacob's ladder. If the suture is not removed, it usually tears through in about six to 10 days. Since this new modified technique and procedure has been adopted, the immediate results appear to be most satisfactory. The progressive diminution of the depression has been observed later.

In older children and adults, the deformity is fixed with a depressed gladiolus and distorted angulated costal cartilages (see Figs. 12 and 14). The gladiolus of the sternum has been pulled in like a door with a hinge at the angle of Ludwig. The deepest point of the depression is at the gladiolar-xiphoid junction. To

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SUMMARY OF INFANTS TREATED BY PHRENOSTERNOLYSIS

*Case 1*, T.B. *Age*, 7 months. *Sex*, Male. *Abnormality First Noticed*, At birth. *Degree of Retraction*, Severe. *Cardiac Signs*, Tachycardia and dyspnea, apical systolic murmur. *EKG*, Not taken. *Operation*, Incision gladiolar-xiphoid junction, 1 cm. lowermost cartilages from sternal junction. Separation of diaphragmatic attachment. Wire traction 5 days. *Post-operative Stay in Hospital*, 9 days. *Result*, Moderate improvement in retraction. The present procedure as described would have given a better result.

*Case 2*, R.V. *Age*, 5 months. *Sex*, Male. *Abnormality First Noticed*, Few weeks old. *Degree of Retraction*, Severe. *Cardiac Signs*, Severe dyspnoea on crying. *EKG*, Inverted T waves in $L_3$ and $L_4$. *Operation*, Phrenosternolysis as described. *Post-operative Stay in Hospital*, 6 days. *Result*, Marked improvement.

*Case 3*, J.McC. *Age*, 2½ years. *Sex*, Female. *Abnormality First Noticed*, At 6 months, but increasing in depth. *Degree of Retraction*, Severe. *Cardiac Signs*, Dyspneic on exertion and during crying spells. *EKG*, Tendency to right axis deviation. *Operation*, Phrenosternolysis as described, wire traction suture 7 days. *Post-operative Stay in Hospital*, 10 days. *Result*, Moderate improvement. A chondrosternoplasty would have given a better cosmetic result.

*Case 4*, J.F. *Age*, 21 months. *Sex*, Female. *Abnormality First Noticed*, Parents unaware until hospitalized for cleft palate and harelip. *Degree of Retraction*, Moderate. *Cardiac Signs*, None. *EKG*, Not taken. *Operation*, Phrenosternolysis as described. *Post-operative Stay in Hospital*, 1 day. *Result*, Marked improvement.

*Case 5*, J.P. *Age*, 12 months. *Sex*, Male. *Abnormality First Noticed*, At birth. Pediatrician advised waiting. *Degree of Retraction*, Severe. *Cardiac Signs*, Heavy breathing with retraction. *EKG*, Not taken. *Operation*, Phrenosternolysis as described. *Post-operative Stay in Hospital*, 5 days. *Result*, Marked improvement.

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correct this deformity various operations have been proposed. The operation most commonly used is that suggested by Brown. This was first performed by the author in 1940. Since the war, the author has modified Brown's procedure and has found the results have been more satisfactory. Chondrosternoplasty, a term suggested by the author for this operation, is indicated in the moderate and severe deformities that are accompanied by cardio-respiratory symptoms which interfere with routine physical activity. Equally important it is indicated in those patients who have developed severe neurotic symptoms and have failed to respond to psychotherapy. Master and Stone[5] state that "re-assurance is usually the only treatment necessary—surgical interference is not indicated except under most exceptional circumstances." In the patients that have received surgery and who are reported below, all have been severely handicapped with the exception of one. In this particular patient (Case 11) who was regarded as psychoneurotic, re-assurance and psychotherapy failed to relieve his symptoms. Since his operation, he is now performing strenuous work without any symptoms.

*Chondrosternoplasty*

The author employs an inverted Y incision beginning at the angle of Ludwig down to the gladiolar-xiphoid junction. At this point the incision turns and runs parallel to the costal margins (Figs. 9 and 11). The pectoral and recti muscles are deflected off the costal cartilages on each side. Beginning usually with the third or fourth cartilages, depending on which is depressed, as much of the perichondrium as possible is stripped off from both surfaces of the cartilage. About one centimeter of the cartilage is removed at the point of angulation. This is repeated down to the fused sixth and seventh cartilages. At this point the xiphoid which is quite depressed is removed. The diaphragmatic edge is freed from its sternal attachment. By means of a hook the sternum is elevated gradually as the pleura is stripped away from the stumps of the cartilages and the undersurface of the sternum by blunt dissection. The pleural cavity will not be entered if this step is performed carefully and slowly, working toward the mid line. The other side is treated in a similar manner (see Fig. 10). A narrow wedge of sternum down to the posterior cortex is incised by means of a motor saw or chisel near the angle of Ludwig where the depression began. After this maneuver, the sternum may be freely elevated to any desired height. When this is done it will be found necessary to trim away more cartilage from the cut ends because of the shorter distance to the raised sternum. A piece of free cartilage is removed from one of the costal margins

of sufficient size to be sutured across to each lowermost cartilage so that the lower end of the gladiolus will rest upon it. The end of the gladiolus is wired to this free cartilage graft. Two wire sutures unite the transected sternum to help maintain its elevated position. The cut edges of the cartilages on each side are united with wire sutures without tension (see Fig. 11). If a pleural cavity is inadvertently entered, the lung is inflated with positive pres-

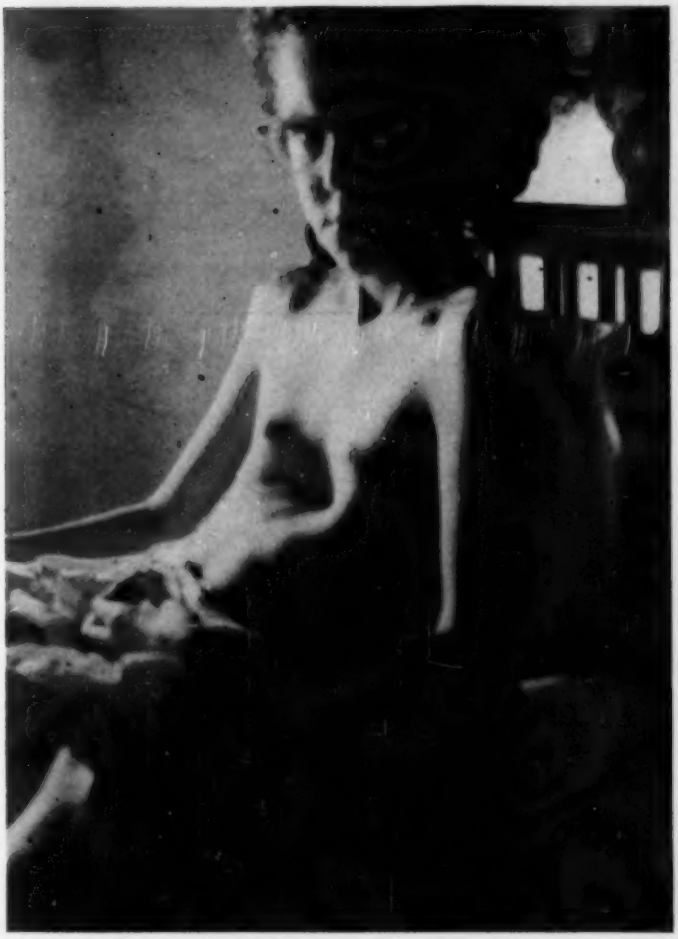

FIGURE 12 (Case 9): Age 10 years. Illustrating a severe case of congenital chondrosternal depression.

sure before complete closure. The muscles are replaced and sutured into position with fine chromic sutures. A rubber tissue drain is placed into the anterior mediastinum and brought out at the lower end of the wound. The skin is sutured with interrupted skin sutures.

Post-operatively it is best to administer oxygen for 24 hours. The patient is permitted out of bed the next day, the drain is removed on the second post-operative day. Since the employment of the free cartilage graft which acts as a scaffold for the elevated sternum, no traction apparatus has been employed. Far better immediate and later cosmetic results have been obtained since the employment of this operative procedure.

### *Reports of Chondrosternoplasty Cases*

*Case 6:* C.R. Female. Age 11, had a severe chondrosternal depression. It was first noticed in infancy. As her activity increased during childhood, it became obvious to the parents that she was thin, tall, underweight, easily

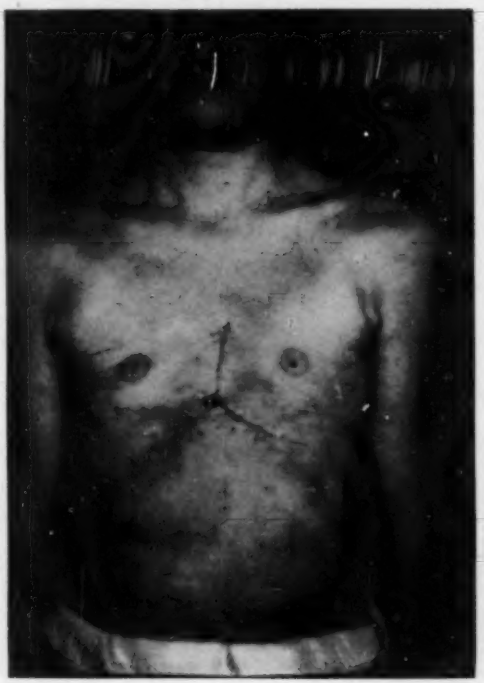

FIGURE 13 (Case 9): Following operation of chondrosternoplasty showing practically a normal chest. Note incision.

fatigued and often fainted on unusual physical exertion. She was referred to the cardiac clinic and treated for years for rheumatic heart disease because of her symptoms and the presence of a loud systolic murmur at the apex and base of the heart. The EKG showed only the low voltage, tachycardia, sinus arrhythmia and deviation of electrical axis. On June 11, 1940 she was operated in the manner described by Brown[3] at the Newark Beth Israel Hospital. On discharge, there was moderate improvement in the chondrosternal depression. The anteroposterior diameter of the thorax was increased by 1 inch. The murmur promptly disappeared. A year later, the mother reported that the child indulged in normal physical activities without discomfort or complaints. The last check-up in 1949 disclosed that she was efficiently working as a sales clerk and had no cardiac symptoms.

*Case 7:* N.M. Male. Age 25 years, had a moderately severe fixed chondrosternal depression which measured 4 cm. in depth at the gladiolar-xiphoid junction. It was first noticed in early infancy. The parents were told that it was due to ricketts and would disappear later in childhood. He was always underweight and never very active in sports or work. He became an artist. He suffered from mild exertional dyspnea and slight but frequent dizzy spells. The most interesting physical finding was an orthostatic pulse and blood pressure. In the standing position the pulse

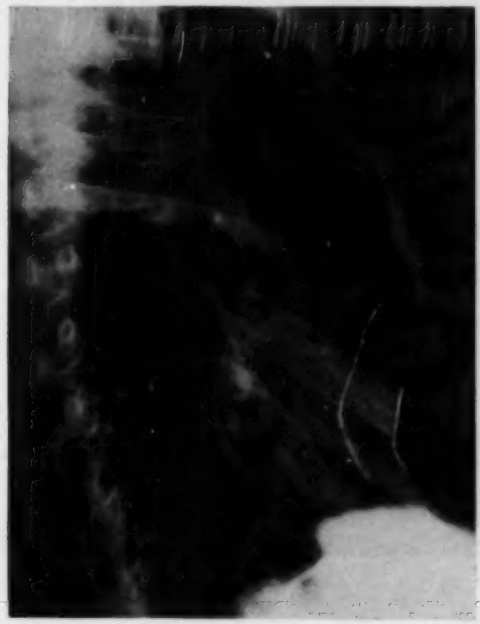

FIGURE 14: Pre-operative lateral x-ray view of Case 9 showing the marked depression of the sternum.

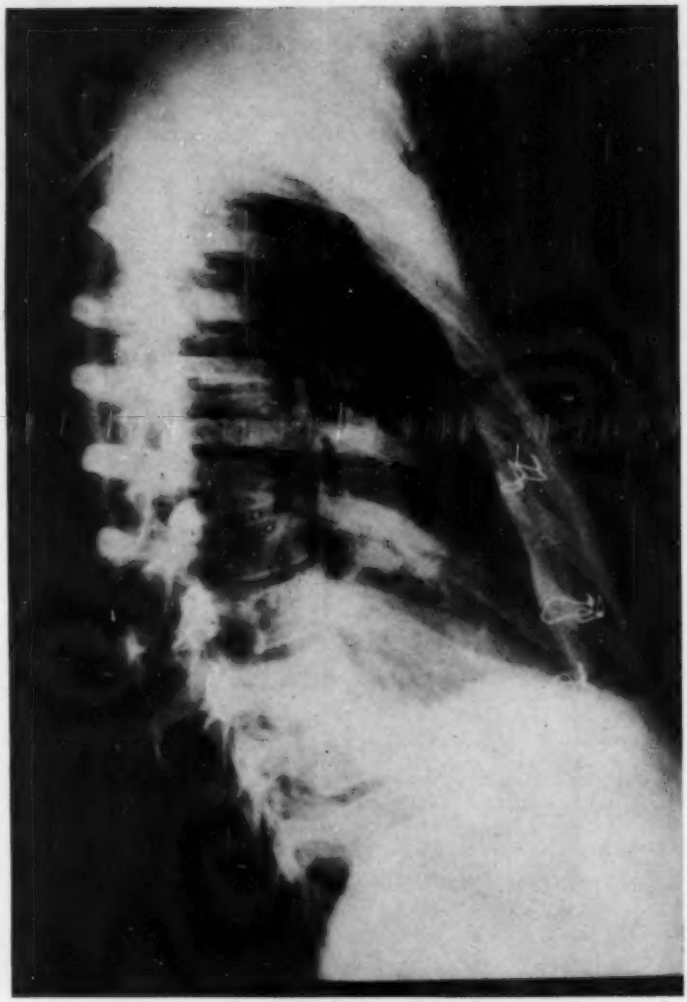

FIGURE 15: Post-operative x-ray view of Case 9 showing the elevation of the wired sternum.

was 105 and the blood pressure 110/88. Immediately on lying down the pulse was 75 and the blood pressure 125/75. This finding has been looked for and found in the last three patients and has proved to be a common finding in this deformity. The EKG showed low voltage; slurred QRS in lead 1; Q wave present and T wave inverted in lead 3; inverted T wave in lead 4. A chondrosternoplasty as described by the author was performed at the Newark Beth Israel Hospital on June 27, 1949. He was discharged after 10 days with a good cosmetic result. The post-operative EKG showed little change. A small stitch abscess was the only untoward development. He has manifested moderate improvement in his ability to indulge in greater physical activity.

*Case 8:* A. M. Male. Age 26, had a severe chondrosternal depression which was noticed at birth. He always tired easily and was short of breath on exertion. He never participated in athletics or could he continue long at an arduous job. During the war he was refused by all services. Physical examination was negative except for the following facts: he was very tall, thin, round shouldered with a severe chondrosternal depression which measured 7 cm. in depth. He had marked orthostatic pulse and blood pressure. On standing the pulse was 144 and the blood pressure was 115/110. Immediately on lying down the pulse dropped to 72 and the blood pressure became 130/80. Circulation time and venous pressure tests were normal. The EKG showed inverted T waves in lead 3 and the post-operative EKG showed little change. A chondrosternoplasty operation as described was performed with an excellent cosmetic result. For the past year he has been engaged in strenuous work without difficulty. There was little change in pulse and blood pressure at the last examination on change of position.

*Case 9.* D.G. Female. Age 10 years, had a severe chondrosternal depression. She was delivered normally and was breast fed until seven months of age. Her parents said that although they noticed her chest was "different" at birth it was not until the infant was one and a half years old that they realized it was abnormal and depressed. They said this depression became progressively more pronounced until the age of five years. The parents were told by their doctor that the deformity was due to ricketts and that the infant would outgrow it. She always complained of pains in the chest, shortness of breath and tiring easily on slight exertion. She was unable to play with children of her age. She became shy and retiring and had a typical posture which is seen with this deformity. The pre-operative EKG report showed a regular sinus rhythm, right axis deviation, tall $T_1$, semi-inverted $T_2$, inverted $T_3$ and $T_4$. Right ventricular strain probably due to anatomical position. A chondrosternoplasty operation as described by the author was performed in January 1949 at the Irvington General Hospital. The immediate cosmetic result was gratifying and there has been marked improvement in her activity (see Figs. 12, 13, 14 and 15).

*Case 10:* M.B. Female. Age 5, had a severe chondrosternal depression which was noted at birth and has increased in size since. She had always been frail and underweight, tired easily at play and had attacks of nocturnal dyspnea and cyanosis. She was considered to be retarded both physically and mentally by her physician. Examination revealed a persistent tachycardia and an orthostatic pulse. On standing it was 150 and on lying 104. The blood pressure readings were too unreliable to report.

There was a systolic murmur audible over the pulmonic area. The x-ray film showed the heart to be entirely in the left thorax. The EKG showed a diaphasic T wave in lead 3. A chondrosternoplasty as described above was performed at the Newark Beth Israel Hospital in July 1949. She was discharged two weeks later with an excellent cosmetic result. Since the operation she has been far more active physically without complaints. The last recent examination disclosed no murmur, a pulse rate of about 90 in both the upright and lying positions.

*Case 11:* C.McC. Male. Age 17 years, had a moderate chondrosternal depression which was noticed two years previously at the age of 15 following marked loss of weight. He was tall and otherwise well developed. In spite of his complaints of tiring quickly and shortness of breath on moderate exertion, the author felt his symptoms were largely psychoneurotic. An effort was made to reassure him and his family and to dissuade them from surgery. They felt that since he had not been able to do strenuous work because of the deformity, they preferred to have the operation performed. Physical examination was negative except for the depression. The pre-operative EKG interpretation was "deeply inverted $T_3$ wave, of configuration to suggest clockwise rotation with right ventricular preponderance. Deep S wave extending to $CF_4$." A chondrosternoplasty operation as described by the author was performed in July 1949 at St. Barnabas Hospital. The post-operative course was uneventful except later he developed a wound infection which drained and healed after several weeks. The post-operative EKG interpretation was "$S_3$ now present. $T_3$ is only shallow inverted. $SCF_4$ still prominent." He now works at a strenuous job without any difficulty.

## SUMMARY

Congenital deformities of the anterior chest wall which are believed by the author to be due to the contractions of a congenital abnormally developed diaphragm have been briefly discussed. Based upon this theory, a new nomenclature and classification have been suggested.

Infants with a congenital chondrosternal depression (funnel chest) have a mobile inspiratory retraction of the chondrosternal area and the epigastrium. This is due to the pull of the congenitally deficient tendinous anterior portion of the diaphragm on its attachment to the chondrogladiolar-xiphoid junction. To offset this mechanism thereby reducing this retraction, and prevent the fixed depression which would develop later, the author prescribes the operation, phrenosternolysis, as early as possible. A description of the operation as practiced by the author is given. Tabulated is a summary of five infants who were treated by phrenosternolysis with satisfactory results.

In childhood, the deformity becomes fixed and comparatively immobile. In the older group with this depression, definite cardiorespiratory symptoms and signs result because of the compression or rotation of the heart and great vessels. In these patients the

cardiorespiratory symptoms as well as emotional disturbances have been severe enough and sufficiently incapacitating to warrant surgical correction. For such patients, the author employs an operation he has termed chondrosternoplasty, the details of which are described. Summaries of the case histories of six patients so treated are given. In all these patients the cosmetic result has been most satisfactory. All have been able to indulge in greater physical activity. The adult group are now performing arduous labor whereas before they could not work at any strenuous job. Attention is invited to the finding of a marked orthostatic pulse and blood pressure in this group.


## RESUMEN

Se han discutido brevemente las deformidades congénitas de la pared torácica anterior que, en la opinión del autor, se deben a las contracciones de un diafragma de desarrollo anormal congénito. Se ha sugerido una nueva nomenclatura y clasificación basadas sobre esta teoría.

Los niñitos con depresión condroesternal congénita (pecho de embudo) tienen una movible retracción inspiratoria de la zona condroesternal y del epigastrio. Eso se debe a que la parte anterior tendinosa del diafragma, congénitamente deficiente, causa tirantez sobre el lugar donde se une con la articulación condrogladiolarxifoidea. Para neutralizar este mecanismo, reducir esta retracción y evitar la depresión fija que se desarrollaría más tarde, el autor aconseja que la operación, la frenoesternolisis, se haga tan pronto como sea posible. Se describe la operación como la ejecuta el autor. Se arregla en forma de tabla los resúmenes de cinco niñitos tratados con la frenoesternolisis con resultados satisfactorios.

En la niñez la deformidad se hace fija y comparativamente inmóvil. En ciertos pacientes con esta depresión se desarrollan síntomas y signos cardiorrespiratorios bien definidos, debidos a la compresión o rotación del corazón y de los grandes vasos. Los síntomas cardiorespiratorios y los desarreglos emocionales en estos pacientes han sido lo suficiente graves e incapacitantes para justificar la corrección quirúrgica. Para estos pacientes el autor emplea una operación que él llama condroesternoplastía, cuyos detalles se describen. Se presentan los resúmenes de los protocolos de seis pacientes tratados en esa forma. El resultado cosmético ha sido muy satisfactorio en todos los pacientes. Todos ellos han podido participar en mayores actividades físicas. Los adultos ahora desempeñan trabajos arduos mientras que anteriormente no podían hacer ningún trabajo estrenuo. Se llama la atención al hallazgo de un pulso y presión sanguínea ortostáticos marcados, asociados con esta deformidad.

## RESUME

L'auteur expose rapidement la question des déformations congénitales de la paroi antérieure du thorax. Il considère que ces altérations sont le résultat des contractions d'un diaphragme congénitalement anormal. Cette théorie sert de base à une nouvelle classification.

Les jeunes enfants qui sont atteints d'une dépression chondrosternale congénitale présentent une rétraction inspiratoire de la région chondrosternale et de l'épigastre. L'origine en est la traction sur la jonction chondro-xiphoidienne, où elle s'insère, de la partie tendineuse antérieure du diaphragme, congénitalement insuffisante.

L'auteur se propose de lutter contre les conséquences de ce mécanisme, de réduire la rétraction et de prévenir la dépression définitive qui pourrait se développer plus tard. Dans ce but, il propose une opération, la phrenosternolyse qui doit être pratiquée aussi rapidement que possible. Il donne la description de son intervention et il résume les observations de 5 nouveaux-nés traités par phréno-sternolyse avec des résultats satisfaisants.

Au cours de la 2ème enfance la déformation se fixe et devient relativement immobile. Chez certains malades atteints de cette dépression on peut voir survenir des troubles cardiorespiratoires dus à la compression ou à la rotation du coeur et des grands vaisseaux. Chez de tels malades, les signes cardio-vasculaires aussi bien que les troubles émotionnels ont été tels que le traitement chirurgical n'a pu être réalisé. C'est alors que l'auteur utilise une intervention qu'il a désignée sous le nom de chondrosternoplastie. Il décrit cette opération en détails et il donne le résumé des observations de six malades traités par ce procédé. Chez tous le résultat esthétique a été tout à fait satisfaisant. Tous ont pu se livrer à une plus grande activité physique. Les adultes sont maintenant capables de fournir un effort pénible alors qu'auparavant ils ne pouvaient même pas se livrer à une activité très modérée. L'auteur appelle la tension artérielle associée à cette déformation.


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# Resection of the Auricular Appendages*

WILLIAM P. LONGMIRE, JR., M.D., JOHN M. BEAL, M.D. and
WILLIAM H. LEAKE, M.D.
Los Angeles, California

It has been stated that rheumatic heart disease is more often responsible for embolic episodes than is any other type of heart disease.[14] As a means of preventing recurrent arterial embolism, resection of the atrial appendages has been proposed.[11]

The frequency of mural thrombi in the auricles of patients with rheumatic heart disease and auricular fibrillation has been noted at autopsy in several studies. Bull[3] in 1921, stated that the majority of peripheral emboli had their origin in the heart, and he recorded five cases of mitral stenosis in which the auricles were the site of formation of thrombi resulting in peripheral embolism. Danzis[5] in studying arterial embolectomy in 1933 said, "The predominating factors in the causation of arterial emboli are affections of the heart and blood vessels . . . 60 per cent of our cases are purely of cardiac origin."

Garvin[6] stated in 1941 that auricular fibrillation bore a highly significant relationship to the occurrence of mural thrombi. In the examination of 116 cases of rheumatic heart disease, mural thrombi were found in 37. Of the 60 patients who had auricular fibrillation, 26 (43.3 per cent) were demonstrated to have mural thrombi, and 86.5 per cent of the thrombi were present in the atria. Other authors also have found a high incidence of thrombi in rheumatic heart disease associated with auricular fibrillation. Graef and his associates[8] found atrial thrombi at autopsy in 11 of 14 patients with this condition. Hay and Levine[9] reported auricular thrombus formation in 49 per cent of 106 cases of rheumatic heart disease with auricular fibrillation. In the report by Graef, in no instance was a thrombus found in the chamber of the auricle proper, and the left auricle was affected more severely than the right.[8]

The frequency of embolic episodes in these cases has been stressed by Garvin,[7] who recorded that in those instances in which

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thrombi were present in the left side of the heart, one or more infarcts were observed in the brain, kidneys, spleen, intestines and/or extremities in 48.7 per cent. Pulmonary infarcts were present in 56.5 per cent of those with thrombi in the right cardiac chambers.

This is supported by the study made by Weiss and Davis of 474 autopsied cases of rheumatic heart disease.[13] In 164, heart disease was the cause of death, and of this group, 73 (45 per cent) were observed to have single or multiple visceral or pulmonary infarcts. Cerebral infarction was described as the cause of death in 22. In the same series, in a group of 28 patients with extensive auricular thrombi, the rhythm was determined in 25. Eighty-eight per cent of these had exhibited auricular fibrillation.

The relatively high morbidity and mortality resulting from peripheral arterial emboli was stressed by McClure and Harkins,[12] who reported a mortality rate of 40 to 59.4 per cent, and amputation because of resulting gangrene in 18 to 22 per cent. The serious nature of peripheral embolism has been more recently stressed by Andrus.[1] Also, this author has indicated the importance of the presence of cardiac failure in the prognosis of peripheral embolism.

Experimental studies suggested that resection of the auricular appendage need not interrupt normal heart action and that it may promote prolonged survival.[2,4,10] In dogs from which the atrial appendage was removed, endothelialization was complete at the site of amputation, and there was no apparent tendency for thrombus formation at the line of suture.

The report by Madden[11] in 1949, indicated that this procedure was feasible in the human being. Stimulated by his report, we performed resections of the auricular appendages in the following cases:

*Case 1:* R.B., a 50 year old white male, gave a history of "heart trouble" following an apparent coronary occlusion in 1938. In succeeding years, he was asymptomatic except for mild substernal discomfort on exertion, and occasional ankle edema.

He was admitted to Wadsworth General Hospital on July 11, 1949, complaining of abdominal pain of one week's duration. The pain was of sudden onset and was accompanied by a transient syncope. Nausea, vomiting and diarrhea with melena followed, lasting three days. The abdominal pain persisted. At the time of admission, he appeared well nourished and developed but chronically ill. Blood pressure was 130/90; pulse 68 and irregular. Neck veins were slightly distended. There was evidence of fluid in the right pleural space. Cardiac dulness extended to the left anterior axillary line in the sixth interspace. Tenderness and muscle guarding were noted in the right lower quadrant of the abdomen, and the liver extended three centimeters below the costal margin. Minimal ankle edema was present.

*Laboratory examination:* The results of the laboratory examination

disclosed a white blood cell count of 14,200, normal hemoglobin and red blood cell count. The Cardiolipin reaction was negative. Blood urea nitrogen was 16 mg. per 100 cc. Stools were negative for gross and occult blood. Roentgenogram of the chest, July 12, 1949, showed right pleural effusion, and fluoroscopic examination indicated mitralization of the heart. Electrocardiograms demonstrated auricular fibrillation and right axis deviation.

*Course:* He improved after the administration of digitoxin, mercuhydrin and a low salt diet was begun. Eleven days following admission he had sudden onset of pain in the left lower extremity, associated with palor and absence of popliteal and tibial pulsations. Three hours following onset of pain, left femoral embolectomy was performed. A similar episode involving the right lower extremity occurred on July 31, 1949, and right femoral embolectomy was performed five and one-half hours later. In both instances the legs remained cold and cyanotic after operation. Three days after the second embolectomy the patient developed pain and numbness of the left arm, which persisted for 48 hours. Digitoxin dosage was increased and refrigeration of the legs was instituted.

Left auricular appendectomy was performed on August 9, 1949. The pathologist reported old and recent organizing mural thrombi (Fig. 1). The course after operation was satisfactory and on August 26, 1949, bilateral mid-thigh amputations were performed. Since then he has received digitoxin daily, a low salt diet and one to two injections of mercuhydrin weekly. There has been no evidence of further embolism.

*Case 2:* W.D., a 63 year old male, had a long history of rheumatic heart disease with mitral stenosis and auricular fibrillation. He had previously

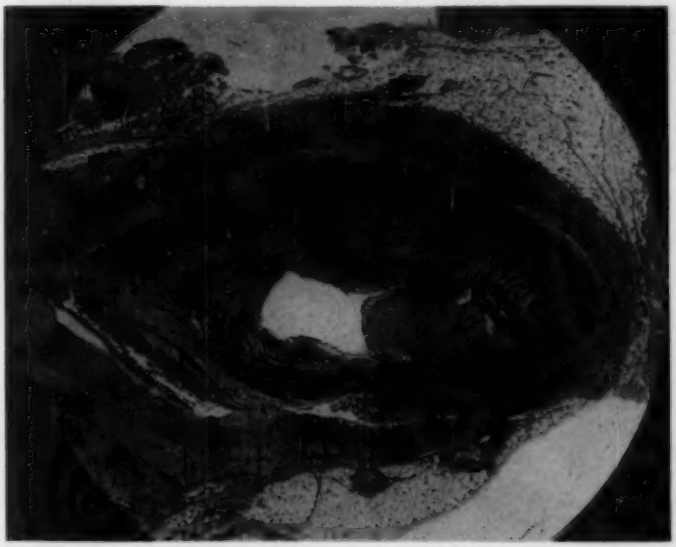

FIGURE 1

required hospitalization for pulmonary infarcts, and in 1947, for a transient left hemiparesis.

He was admitted to Wadsworth General Hospital on November 5, 1949, because of the sudden onset of right chest pain and bloody sputum of two days' duration. Examination disclosed a thin, chronically ill, white male. Bright red sputum was noted. Moderate dyspnea was present. Blood pressure was 148/86; apical pulse about 84, irregular with a pulse deficit of about 20. Breath sounds were diminished at the right base posteriorly and moist rales were heard at both bases. Cardiac dulness was enlarged both to right and left. A systolic and a diastolic murmur were present at the apex. Liver edge was 4 cm. below the costal margin.

*Laboratory examination:* Serological test was negative, red blood cell count and hemoglobin normal, and white blood cell count 10,600. Urinalysis was negative. Roentgenogram of chest demonstrated cardiac enlargement, pulmonary emphysema and a shadow in the right lower lung field, which was interpreted as pulmonary edema with effusion. Electrocardiogram showed auricular fibrillation and the effects of digitalis.

*Course:* When the patient was treated with bed rest and general supportive measures, the amount of hemoptysis decreased until two weeks after admission. At this time a sudden episode of chest pain occurred, associated with hemoptysis, and was followed a week later by a similar episode. No evidence of thrombophlebitis was observed on repeated examinations of the lower extremities.

Right auricular appendectomy was performed on December 16, 1949. An organizing mural thrombus was reported by the pathologist (Fig. 2). The patient recovered satisfactorily. In an effort to determine whether the veins of the lower extremity were the sites of origin of the pulmonary

FIGURE 2

emboli, bilateral superficial femoral vein ligations were performed on January 11, 1950. Exploration of the distal venous system disclosed no evidence of thrombus formation. The patient subsequently has been maintained on a cardiac regime and has had no further embolic episodes.

*Case 3: H.B.*, a 53 year old retired elevator operator, had rheumatic fever at the age of 23. He was unaware of cardiac involvement until 1947. At this time, sudden severe pain in the left leg necessitated his hospitalization elsewhere. He recovered after anti-coagulant therapy was begun. At that time, he was found to have auricular fibrillation and was given digitalis, which he continued to take until the date of the present admission. A similar episode of pain occurred in 1948, followed by intermittent claudication. He was hospitalized in April, 1949, for pulmonary embolus. During the same admission left lumbar sympathectomy was performed. In October, 1949, a transient episode of pain in the right leg occurred, followed a few weeks later by a sudden attack of right lower quadrant pain of six hours' duration. Occasional dyspnea and ankle edema had been noted for 14 years.

He was admitted to Wadsworth General Hospital on January 10, 1950,

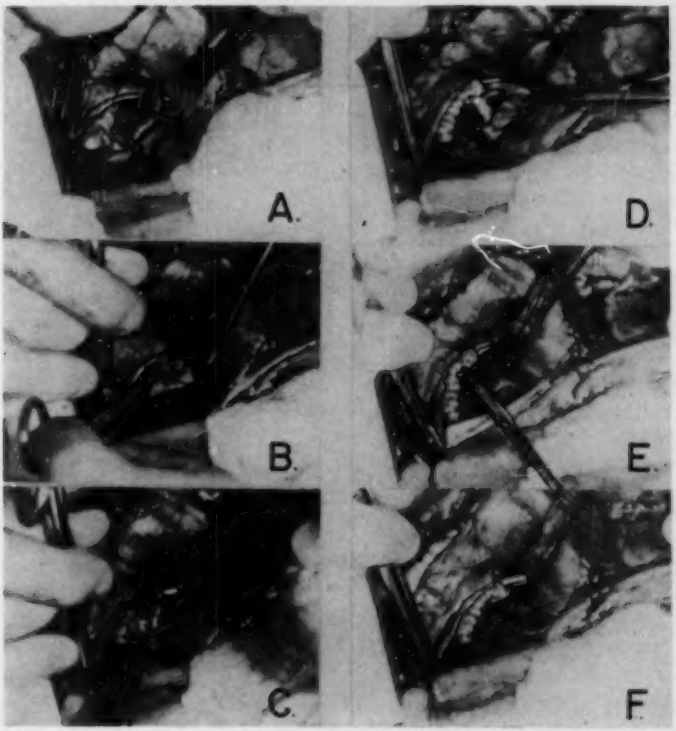


FIGURE 3

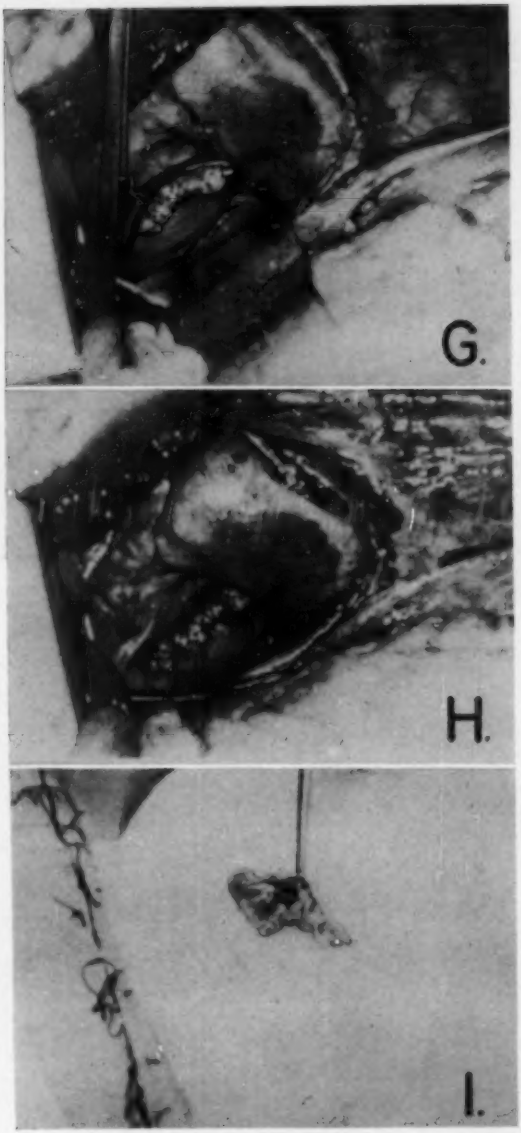


FIGURE 3 (Continued)

complaining of pain and numbness of the right hand of four hours' duration. The onset of the pain was sudden and the attack was associated with palor of the right hand and forearm. These symptoms spontaneously subsided.

*Examination:* The patient was a thin, white male. Blood pressure was 120/70; apical pulse rate 80, irregular, with a pulse deficit of 10. Lung fields were clear. The heart was enlarged to percussion and no murmurs were detected. Peripheral vessels were sclerotic and dorsalis pedis pulses were absent bilaterally. The right hand was dusky and cooler than the left, but radial and ulnar pulses were present and equal bilaterally.

*Labratory reports:* Urine analysis was negative; blood count normal except for white blood cell count of 11,000. Kahn reaction was positive (8 units); Wassermann negative. Roentgenogram of chest showed cardiac enlargement and chronic pulmonary congestion. Electrocardiogram demonstrated auricular fibrillation with a ventricular rate of about 60 and occasional extrasystoles.

*Course:* The patient was heparinized for 48 hours and showed marked improvement. Left auricular appendectomy was performed on January 17, 1950. A thrombus was demonstrated in the appendage at operation (Fig. 3). The postoperative course was satisfactory. Digitalis administration and low salt intake have been continued. No further evidence of embolism has been noted.

### *Operative Procedure*

For the operations performed on the patients in this series, endotracheal ether anesthesia was used following induction with intravenous sodium pentothal. Narcotics were used in minimal amounts in pre-operative medication to prevent respiratory depression.

The operative procedure was as follows: The auricle was approached through an anterior incision in the third interspace. The pericardium was incised anterior and parallel to the phrenic nerve. A special non-crushing right angle clamp was placed across the base of the appendage and a continuous No. 0000 silk suture placed distal to the clamp. The appendage was partially excised distal to the suture and a second continuous suture of No. 0000 silk used to approximate the edges of the exposed cuff. The appendage was then excised entirely as the suture line progressed. Partial release of the clamp was followed in these cases by some bleeding along the line of suture. The insertion of a few additional mattress sutures of silk controlled the bleeding, after which the clamp was removed completely. The pericardium was closed with interrupted sutures and a small opening left in the pericardial closure to prevent tamponade. The chest was closed without drainage.

*Subsequent course:* The three patients (Fig. 4) have survived operation and are at the present time alive. In the first case (R.B.) bilateral mid-thigh amputation was required because of the occurrence of gangrene in the lower extremities. In each case

digitalis administration and low salt intake have been continued. There has been no evidence of further embolism.

Electrocardiograms obtained after removal of the auricular appendage in these patients demonstrated no significant changes when compared with the tracing made prior to operation.

*Discussion*

From the evidence obtained at post-mortem examinations, it was indicated that the majority of peripheral emboli in patients with rheumatic heart disease and persistent auricular fibrillation originate in the auricular appendages. Removal of the auricular appendage, which can be accomplished in man with reasonable safety, seems to be one logical approach to the prevention of these serious vascular accidents.

Before surgical intervention, attempts at conversion to normal sinus rhythm were made in each of the patients in this series without success. This procedure should be considered prior to operation in similar cases; however it was noted in the studies already mentioned that there is a significant incidence of mural thrombus formation in the auricles of patients who have rheumatic heart disease and mitral stenosis unaccompanied by auricular fibrillation.

The use of anti-coagulants as a means of preventing formation or discharge of mural thrombi is worthy of consideration. Therapy by this means would require prolonged administration and careful supervision and, at times, the use of these drugs may be hazardous.

Criteria for selection of patients for resection of the auricular

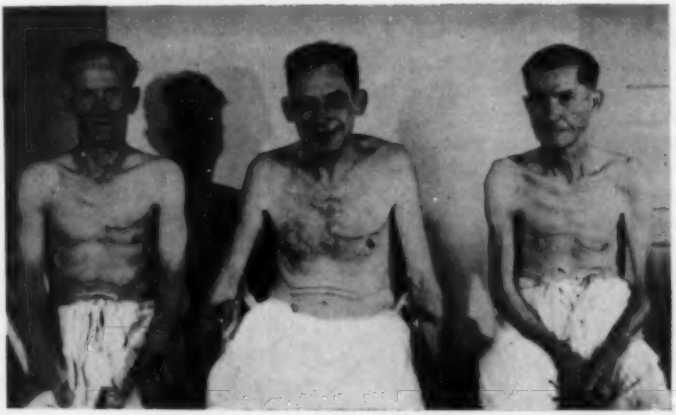

FIGURE 4

appendage cannot be established on the basis of our present limited experience. The decision for operative intervention in this series was made after consultation between the cardiologists and the surgical staff. Close cooperation between the cardiac and surgical services is required for the supervision of the patients' postoperative care, as well as for their evaluation as surgical possibilities before operation.

## SUMMARY

Resection of the auricular appendage for recurrent embolism was performed in three cases of rheumatic heart disease with persistent auricular fibrillation.

Mural thrombi were demonstrated in each case. Cardiac function was not apparently influenced by the operative procedure. No evidence of further embolization has been noted over a follow-up period of five to 10 months.

## RESUMEN

Se llevó a cabo la resección del apéndice auricular por embolias repetidas en tres caos de enfermedad cardiaca reumática con fibrilación auricular persistente.

En todo caso se demostraron trombos en las paredes. Aparentemente el procedimiento quirúrgico no afectó la función cardiaca. No ha habido signos de más embolias en un periodo de observación subsecuente de 5 a 10 meses.

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# Discussion

JOHN F. BRIGGS, M.D., F.C.C.P.
St. Paul, Minnesota

Dr. Beal and his associates together with Dr. Madden and Dr. Dock have presented an excellent thesis in support of the resection of the left auricular appendage to prevent peripheral embolic phenomena in mitral heart disease.

At Ancker Hospital in St. Paul, Minnesota, Dr. Ivan Baronofsky has approached the problem in a similar manner. Rather than resecting the auricular appendage, however, he ligated it at its origin in the left auricle. To date he has performed this procedure on three patients, and although the time limit is short, there has not been any further peripheral embolization. One patient upon whom he operated died of an illness not related to the cardiac disease. At necropsy the left auricular appendage so ligated was almost completely atrophied.

At Ancker Hospital we have been disappointed to some extent in the actual lack of detailed statistics concerning the location of various thrombi in the auricles and auricular appendages in individuals suffering from mitral stenosis. It was because of this that Dr. Theim (in a paper to be published elsewhere) reviewed all of the deaths from mitral stenosis that had come to autopsy at Ancker Hospital. He collected data on a large series of such patients, and we were surprised to find that the thromboses occurred almost equally in both the right and the left auricular appendage. It was also apparent that thrombi were present in the left auricle and in other places within the heart chambers. This has been disturbing to us because it necessitates our being able to determine in what patients the thrombus that has mothered the peripheral emboli is solely located in the left auricular appendage. As it stands now one has no such means of making a definitive localization of the offending thrombus. Further, good results have been reported in preventing further embolization through the use of prolonged anti-coagulant therapy.

Just recently Dr. Baronofsky resected the left auricular appendage as a means of entering the left auricle to perform a commissurotomy on the mitral valve. This operation was performed successfully and the pathologist, Dr. John Noble, reported the presence of thrombi in this auricular appendage. Dr. Baronofsky believes that this combination of procedure should be attempted in order that one remove the possible offending thrombi in the left auricular appendage at the same time that one does the commissurotomy.

Although at the present time our knowledge concerning the value of surgery on the auricular appendage is still in the state of flux, it still goes without saying, however, that under proper circumstances such surgery should be continued for it is only through our medical and surgical experiences that we will ultimately arrive at some conclusion as to how to select cases for this procedure.

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H. BRODIE STEPHENS, M.D.
San Francisco, California

Doctors Longmire, Beal and Leake are to be congratulated upon their excellent results in three patients that were subjected to resection of the auricular appendage. The fact that no recurrent embolism has occurred in the follow-up period is extremely gratifying.

I find there are no statements by the authors upon which I am in disagreement. Emphasis might be placed on the statement in the manuscript that points out that the selection of patients requires individual consideration in each case, and that the decision for operative intervention is made by consultation between the internist and the surgeon. The authors go on to state that further experience and a longer follow-up study is necessary before definite criteria can be established.

I asked Doctor Maurice Sokolow, a cardiologist at the University of California Medical School here in San Francisco, to review this manuscript of Doctors Longmire, Beal and Leake. Doctor Sokolow commended the high quality of the authors' presentation. It has been Doctor Sokolow's experience that he can convert 80 per cent of the patients that suffer from chronic auricular fibrillation by the proper dosage of quinidine to maintain a constant high blood quinidine level. On the other hand, Doctor Sokolow admits the difficulty in preventing relapses from regularity of rhythm. In addition, he tells me that successful series of patients have been reported by Wright and another series by Nichols that have been carried along successfully on long-range anticoagulant therapy with Dicumarol. It was Doctor Sokolow's opinion that most cardiologists today would manage auricular fibrillation accompanied by embolic phenomena by proper quinidine dosage and anticoagulants. Resection of the auricular appendages as described by the authors would certainly seem to be indicated when normal cardiac rhythm cannot be maintained, or in areas where blood quinidine levels and prothrombin time are not available. The authors make no comment on the use of procaine before entering

the pericardial sac. I would appreciate hearing from Doctor Beal in this regard when he closes the discussion. It has been my experience and that of many others that the dog's heart is particularly prone to develop ventricular fibrillation when a clamp is applied to various areas of the heart. Carter states in a recent communication that procaine has been striking in its effect on abolishing irregularities in the dog's heart. The procaine is administered over the surface of the heart and into the auricular appendage.

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## DISCUSSION FOLLOWING PRESENTATION

Dr. Beal (closing): I want to thank the discussants for their pertinent comments. Dr. Briggs has emphasized several important aspects of the problem, and we agree with him that the solution is not clear. We chose the surgical approach because it seemed unlikely that one clinic could obtain sufficient case material to permit evaluation of two approaches at the same time. While the results are gratifying now, it is too early to draw conclusions as to the final outcome. In reference to the question of conversion to normal sinus rhythm, this was unsuccessfully attempted in all three patients in this series. It would appear that conversion should be attempted in most cases before the decision is made in favor of surgical intervention. It is interesting that during the first operation, it was thought that the patient had reverted to normal sinus rhythm during the induction of anesthesia; however, electrocardiograms and exposure of the heart showed this not to be the case. It is to be emphasized again that more information is needed concerning the incidence and location of mural thrombi in the chambers of the heart. Furthermore, additional experience with resection of the atrial appendages is needed before conclusions can be drawn as to the value of this procedure.

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# Tuberculous Meningitis in Children Treated with Combined Streptomycin and Potassium Iodide*

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The treatment of tuberculous meningitis in children less than four years of age with streptomycin in this hospital has been discouraging in that of seven consecutive patients so treated died, while five adults treated in a similar manner are alive one to two years after onset of the disease. The idea of combined potassium iodide-streptomycin therapy for tuberculous meninigtis was brought to our attention by Dr. William J. Kerr of San Francisco, California, who visited our hospital as a consultant in internal medicine for the Far East Command from the Surgeon General's Office in December 1948. Due to the paucity of information, we were hesitant in using this method although an autolytic effect of iodide on caseous material was reported by Jobling and Petersen in 1914.[1] However, recently favorable results were reported from the use of potassium iodide and streptomycin against established tuberculosis in guinea pigs with no evident alteration by potassium iodide on the potency of streptomycin.[2] Early in 1949 an editorial in the Journal of the American Medical Association stated this procedure was being given a clinical trial in tuberculous patients.[3] Emboldened by these reports and working on the idea that there was nothing to lose when confronted with so fatal a disease, we decided to use combined streptomycin and potassium iodide therapy on succeeding cases of tuberculous meningitis among children in this hospital.

This report concerns three consecutive cases treated with combined streptomycin and saturated solution of potassium iodide. In each case tubercle bacilli were found in a direct smear of the spinal fluid, its culture or in the inoculated guinea pig.

*Case 1* is a 14 months old female who was admitted on May 23, 1949 with fever, restlessness and weakness of left leg of two weeks duration. There was no history of stiffness of the neck. The family and past history was negative for pulmonary tuberculosis.

On admission, she was poorly nourished, febrile and acutely ill. The anterior fontanelle was large but not bulging. Lungs were clear and the heart was normal. There was hyperextension of both ankles. The left leg was weak and immobile but she could flex and extend the foot and great

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toe. There was no apparent difference in the size of the legs. Brudzinski and Kernig signs were present with positive Babinski on both sides. The patellar and abdominal reflexes were exaggerated on the right side. The spinal fluid examinations revealed typical findings as shown in Table I.

A pellicle was found in the initial spinal fluid obtained and a guinea pig was inoculated. This animal developed tuberculosis. The blood findings were: white blood cells 14,000, neutrophils 42, lymphocytes 58. The chest x-ray film showed a density in the right upper lobe which was read as a primary lesion. The x-ray film of the skull demonstrated a marked separation of the cranial sutures which was thought to be due to the increased intra-cranial pressure. A diagnosis of tuberculous meningitis was made and she was immediately treated with streptomycin ½ gram every 12 hours intramuscularly, 0.025 grams intrathecally daily and saturated potassium iodide solution, 10 drops daily per orem. She continued to run a low grade fever until the last 30 days of her life when the temperature rose occasionally to 103-104 degrees F. She received a total dose of 25.6 grams streptomycin intramuscularly and .35 grams intrathecally and 38 cc. of potassium iodide. The patient died on September 1, 1949 from a gradually deepening stupor and unconsciousness which persisted in the last 10 of the 108 days of her illness.

The post-mortem examination revealed tubercles involving the brain, basal ganglia, spinal nerve roots and the meninges from which tubercle bacilli were recovered. A primary complex, consisting of a focal caseous lesion and a caseous hilar lymphadenitis was found in the right upper lobe of the lungs.

*Case 2* is a four year old female who was admitted on March 17, 1949 because of fever and vomiting. The illness developed gradually 10 days before admission as a mild headache. The temperature became elevated

TABLE I

| Date | Total Cells | Neutrophils | Lymphocytes | Sugar | Chlorides | Total Proteins |
|---|---|---|---|---|---|---|
| 23 May 49 | 123 | 58 | 65 | 17.0 | 625 | ... |
| 26 May 49 | 214 | 48 | 166 | 20.0 | 627 | ... |
| 27 May 49 | 42 | 3 | 39 | 36.5 | ... | ... |
| 31 May 49 | 53 | 4 | 49 | 18.0 | ... | ... |
| 6 Jun. 49 | 76 | 4 | 72 | 18.3 | 667 | ... |
| 13 Jun. 49 | 175 | 20 | 155 | 21.9 | ... | ... |
| 24 Jun. 49 | 70 | 25 | 45 | ... | 625 | 1,080 |
| 6 Jul. 49 | 210 | 60 | 150 | 32.2 | 585 | 285 |
| 11 Jul. 49 | 226 | 57 | 169 | 23.0 | 521 | 760 |
| 20 Jul. 49 | 235 | 35 | 200 | 25.0 | 627 | 599 |
| 25 Jul. 49 | 163 | 28 | 135 | 26.3 | 584 | 630 |
| 1 Aug. 49 | 171 | 21 | 150 | 22.5 | 626 | 568 |
| 16 Aug. 49 | 105 | 15 | 90 | 39.4 | 646 | 430 |
| 23 Aug. 49 | 58 | 8 | 50 | 27.7 | 625 | 323 |

and vomiting appeared two days afterwards. The vomiting became projectile and with increased frequency on the third day. Weakness was notable. A few days later, slight rigidity of the neck was noticed and on the day before admission she had convulsions followed by unconsciousness for three minutes. She had "asthma" a year before admission. Last attack was in June 1948. Both parents and two sisters are apparently healthy.

On admission she had fever of 104 degrees F. and appeared stuporous. There was marked nuchal rigidity with hyperactive tendon reflexes and positive Kernig and Brudzinski signs. The lungs and heart were apparently normal. Blood examination showed white blood cells 12,400, neutrophils 58, and lymphocytes 39. The spinal fluid examination showed white blood cells 391, neutrophils 190, lymphocytes 201, sugar 37, proteins 268, and chlorides 626. A pellicle was present. The chest x-ray film taken on admission showed evidence of calcified lesions in the apex of the right lung. A diagnosis of tuberculous meningitis was made and she was given streptomycin ⅛ gram (0.12 gram) twice a day intramuscularly; intrathecal streptomycin 0.025 daily for two weeks; and saturated potassium iodide solution 10 drops daily was given by mouth. From the day of admission on March 17 to May 3, 1949 the child had elevated temperatures from 101 to 103 degrees F. almost daily. Rigidity of the neck was always present, she showed practically no improvement during this period. The serial spinal fluid examinations revealed high cell count and low chlorides and sugar as shown in table II.

The clinical course from May till the first week of July was favorable. The temperature became lower and she began to show definite clinical improvement. The child became mre alert and began to talk. Nuchal rigidity was very slight. Appetite improved and vomiting ceased. On July 13, 1949 the streptomycin and potassium iodide therapy was discontinued after a total of 42 grams streptomycin and 78 cc. of potassium iodide had been administered.

Since July the temperature gradually fell to normal and she became active and playful. Rigidity of the neck was practically gone and the reflexes were no longer active. On August 1, 1949 the guinea pig and culture examinations were reported positive for tubercle bacilli. She became asymptomatic from the middle of August except for the spinal fluid examination on August 17 which showed white blood cells 31, neutrophils 6, lymphocytes 25, sugar 40, proteins 597 and chlorides 646. Because of the marked clinical improvement and the insistence of the parents, she was discharged from the hospital on September 15, 1949 as a case of arrested tuberculous meningitis and up to the present date she is known to be well.

*Case 3* is a three year old male who was admitted on May 11, 1949 because of fever, vomiting and rigidity of neck and back. The illness developed a week prior to admission as fever which persisted and made the patient apathetic. Vomiting ensued four days after the onset followed by rigidity of the neck. Previous personal and family history were noncontributory.

On admission he was stuporous and highly febrile. The neck and back were markedly rigid with positive Kernig and Brudzinski signs. The reflexes were hyperactive with ankle clonus present and sustained on both sides. Cremasteric and abdominal reflexes were normal. Blood examination revealed white blood cells 13,000, neutrophils 28, lymphocytes 70, eosinophils 2. The lumbar puncture showed a clear spinal fluid under

increased pressure and white blood cells 134, sugar 17, proteins 213, chlorides 625. Chest x-ray film showed evidence of calcification in the left lung. These findings were suggestive of tuberculous meningitis and immediately he was placed on streptomycin 0.25 grams intrathecally twice a week, and 0.125 grams tid intramuscularly and 10 drops of saturated potassium iodide daily.

He showed no clinical change in the first 60 days. Fever remained high, 102 to 103 degrees F. almost daily. He remained stuporous with marked nuchal rigidity. Vomiting, however, ceased although the reflexes remained hyperactive. In the second week of July definite clinical improvement was noticed. Fever became low; he was more alert and could utter monosyllabic words. The reflexes were still hyperactive but with less nuchal rigidity. Ankle clonus was present but no longer sustained. At this time the guinea pig and culture examinations were reported positive for tubercle bacilli. Table III shows results of the spinal fluid examinations

In August he became fully conscious, coherent and playful with occasional temperatures of 101 degrees F. The neck was only slightly rigid. The serial chest x-ray inspections showed no apparent change. Because of the marked clinical improvement, the streptomycin and potassium iodide therapy was discontinued on October 10, 1949 after he had re-

TABLE II

| Date | Total Cells | Neutrophils | Lymphocytes | Sugar | Chlorides | Total Proteins |
|---|---|---|---|---|---|---|
| 18 Mar. 49 | 391 | 190 | 201 | 37.0 | 625 | 268 |
| 19 Mar. 49 | 190 | 95 | 95 | ... | 656 | ... |
| 21 Mar. 49 | 190 | 90 | 100 | ... | ... | ... |
| 25 Mar. 49 | 253 | 100 | 153 | ... | ... | ... |
| 28 Mar. 49 | 255 | 110 | 145 | ... | 583 | 308 |
| 30 Mar. 49 | 352 | 150 | 252 | ... | ... | ... |
| 1 Apr. 49 | 61 | 7 | 54 | ... | ... | ... |
| 4 Apr. 49 | 218 | 100 | 118 | 23.0 | 625 | ... |
| 7 Apr. 49 | 317 | 100 | 217 | ... | ... | ... |
| 12 Apr. 49 | 360 | 100 | 260 | ... | ... | ... |
| 16 Apr. 49 | 325 | 140 | 185 | 56.0 | 708 | 480 |
| 22 Apr. 49 | 545 | 181 | 364 | ... | ... | ... |
| 26 Apr. 49 | 331 | 160 | 171 | ... | ... | ... |
| 3 May 49 | 26 | 10 | 16 | ... | ... | 806 |
| 28 May 49 | 180 | 11 | 179 | 33.0 | 604 | ... |
| 1 Jun. 49 | 88 | 4 | 84 | ... | ... | ... |
| 16 Jun. 49 | 70 | 5 | 65 | ... | ... | ... |
| 1 Jul. 49 | 94 | 23 | 71 | ... | ... | 108 |
| 17 Aug. 49 | 31 | 6 | 25 | 40.0 | 646 | 597 |

ceived a total of 54 grams of streptomycin and 50 cc. of potassium iodide. However, in the latter part of October the temperature again became unstable with frequent elevations to 100 degrees F. He started to have occasional vomiting spells and lost four pounds within two weeks. This episode was considered a relapse and on November 5, 1949 streptomycin was again given 0.25 gram twice daily intramuscularly with 10 drops saturated potassium iodide daily by mouth. The spinal fluid on December 2, 1949 showed acid-fast bacilli in the smear of the pellicle. He has received a total of 75.25 grams of streptomycin on February 3, 1950.

Since then the child showed occasional temperatures of 100 degrees F.; was irritable and often cried, complaining of pain in the neck. In the past two weeks, he developed a tortion of the neck to the right and a slight right scoliosis. At present there is still a moderate nuchal rigidity with reflexes hyperactive. Ankle clonus is present on both sides and sustained on right only. Babinski is positive bilaterally. Cremasteric and abdominal reflexes are normal. The child is alert and playful when not irritated. The prognosis in this case is still uncertain.

### *Discussion*

We make no pretention of drawing any conclusion on the curative effect of combined streptomycin and potassium iodide treatment in tuberculous meningitis. However, we wish to point out the interesting facts in our series of 10 cases. The first seven were treated with streptomycin alone and the average duration of the disease from the onset to its fatal termination was 18 days. This approximates the 19½ days duration of tuberculous meningitis reported by Lincoln.[4] The last three patients were treated with combined streptomycin and potassium iodide. In Case 1, the disease lasted 108 days from the onset to its fatal termination. In Case 2, the disease developed in the second week of March 1949

TABLE III

| Date | Total Cells | Neutrophils | Lymphocytes | Sugar | Chlorides | Total Proteins |
|---|---|---|---|---|---|---|
| 11 May 49 | 134 | 60 | 74 | 17.0 | 625 | 213 |
| 16 May 49 | 389 | 186 | 203 | 18.0 | 625 | 130 |
| 24 May 49 | 360 | 176 | 184 | ... | 640 | ... |
| 14 Jun. 49 | 148 | 17 | 131 | 15.0 | ... | ... |
| 12 Jul. 49 | ... | ... | ... | 31.0 | 683 | 346 |
| 26 Jul. 49 | 280 | 50 | 230 | 25.8 | 625 | 374 |
| 16 Aug. 49 | 188 | 35 | 153 | 35.0 | 584 | 360 |
| 23 Aug. 49 | 342 | 42 | 300 | 40.0 | 604 | 320 |
| 18 Oct. 49 | 388 | 88 | 300 | 26.0 | 583 | 1,320 |
| 4 Nov. 49 | 605 | 82 | 523 | 33.0 | 580 | 2,910 |
| 2 Dec. 49 | 207 | 51 | 156 | 17.8 | ... | 6,000 |

and the patient was discharged as an arrested case on September 15, 1949 and is still doing well in her home. In Case 3, even though the prognosis is in the balance, the patient is still alive after nine months of the disease.

Merely on the basis of having altered the course of the disease in the three cases reported, we are encouraged to continue and to advocate the use of combined streptomycin and potassium iodide in the treatment of tuberculous meningitis.

## SUMMARY

1) Three children with tuberculous meningitis were treated with combined streptomycin and potassium iodide. The first died 108 days after onset of disease, the second attained a state of arrest and is still alive and in good health 11 months after onset of symptoms, and the third, although the prognosis remains uncertain, is still alive after nine months of the disease.

2) Seven other children with tuberculous meningitis treated with streptomycin alone died on an average duration of 18 days from the onset of the disease.

3) The use of streptomycin and potassium iodide in tuberculous meningitis is recommended as deserving further clinical trial.

## RESUMEN

1) Se trató a tres niños con meningitis tuberculosa con estreptomicina y yoduro de potasio combinados. El primero murió 108 días después del comienzo de la enfermedad, el segundo llegó a un estado de estacionamiento de la enfermedad y todavía vive y continúa en buena salud 11 meses después del comienzo de los síntomas y, el tercero, aunque el pronóstico continúa incierto, todavía vive después de nueve meses de la enfermedad.

2) Otros siete niños con meningitis tuberculosa, tratados con estreptomicina sola, murieron con un promedio de sobrevivencia de 18 días después del comienzo de la enfermedad.

3) La estreptomicina y el yoduro de potasio merecen un ensayo clínico adicional en la meningitis tuberculosa y, por esta razón, se recomienda su empleo.

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# Atypical Pulmonary Inflammatory Reactions

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In the course of routine postmortem microscopic examinations of the lungs of persons, in whom pneumonia had or had not been evident clinically, a number of atypical inflammatory reactions have been observed. The purpose of this report is to describe the changes in a series of 10 cases and to compare them with those in primary atypical pneumonia and in rheumatic pneumonia.

## *Clinical and Pathological Data*

*Case 1:* A white machinist, aged 45, had a fatal illness of seven days' duration. Clinical diagnoses were: coronary thrombosis, with myocardial infarction, and extensive bilateral bronchopneumonia. He became ill suddenly with nausea, shortness of breath, and general weakness, followed within two days by pedal edema, cough, abundant bloody sputum, and pain in the chest. Temperature was 101 degrees F., pulse 134, respirations 44, and blood pressure 140/116. He was extremely cyanotic and dyspneic. Both lungs were patchily dull and revealed moist rales and bronchial breath sounds. The heart was slightly enlarged. X-ray film inspection showed extensive mottling of both lung fields. Electrocardiogram indicated recent anterior left-ventricular infarction. The white count was 18,750 with 90 per cent neutrophils. Necropsy revealed an incompletely-obstructive thrombus in the anterior descending branch of the left coronary artery, with early left-ventricular infarction and fibrinous pericarditis, and extensive bilateral bronchopneumonia. The right and left lungs weighed 1585 and 1625 grams respectively, were congested, and extensively nonaerated. Microscopically, the alveoli contained mononuclear cells, inspissated fibrin, hyaline membranes, and frequently proliferated septal cells. Buds of fibrin and entangled mononuclear cells projected into the lumina of alveoli and of alveolar sacs and ducts. Some smaller arteries showed mural fibrinoid swelling. Scattered subiobular areas contained neutrophils, some of which were necrotic. Histologic diagnosis was bilateral atypical pneumonia.

*Case 2:* A white woman, aged 56, who had been hospitalized for pneumonia and influenza twice within four years, had chronic myelogenous leukemia and died within a few hours of her third hospital admission. Clinical diagnosis was bronchopneumonia. No laboratory studies were made. Necropsy revealed partial fibrous obliteration of the left pleural cavity, mild coronary sclerosis, cardiac hypertrophy, hepato- and splenomegaly, and bronchopneumonia. The right lung weighed 520 and the left, 610 grams. Both lower lobes exhibited nodular consolidated areas, which were moist, reddish brown, and friable on section. The smaller

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bronchi had thickened walls. Microscopically, the alveoli contained granular precipitate, monocytes, foamy macrophages, and focally-proliferated septal cells. In some areas, the exudate was partially organized by fibroblasts. The hyperemic alveolar walls were infiltrated by monocytes. Focally, edematous bronchial walls were infiltrated by neutrophils and immature leukocytes. A number of bronchioles showed partial absence of the epithelial lining and presence of a granulation tissue that was rich in fibroblasts and lymphocytes and subtotally obliterated the lumen. The histologic diagnosis was atypical pneumonia.

*Case 3:* A white housewife, aged 28, was hospitalized because of progressive weakness and dyspnea, with cough and cyanosis. These symptoms developed during the six days following the premature delivery of twins, accompanied by severe postpartum hemorrhage. The woman had severe secondary anemia and pneumonia of the left upper lobe. Upon hospitalization, her temperature was 98.6 degrees F., pulse 116, respirations 32, and blood pressure 112/82. She appeared acutely ill, pale, edematous, and dyspneic. Both lungs revealed coarse rales. The heart, enlarged to the left, exhibited a systolic apical murmur. The liver was enlarged, as was the uterus, which still bled. Hemoglobin was 5 grams, erythrocytes were 1.4 million, and leukocytes, 16,600 with 81 per cent neutrophils. Necropsy, following death less than 12 hours after hospitalization, confirmed the clinical diagnoses. The right lung weighed 790, and the left, 775 grams; both were crepitant and contained a moderate amount of bloody frothy fluid. On section, hyperemic and consolidated zones appeared poorly demarcated. Microscopically, scattered areas contained macrophages, hyaline membranes, and swollen septal cells. Some alveoli, sacs, and ducts contained granulomas, composed of fibrin and monocytes, resembling Masson bodies. Other areas were edematous and hyperemic. Occasional arterioles exhibited fibrinoid swelling. Scattered lobules contained exudate composed of fibrin, neutrophils, lymphocytes, and macrophages. The diagnosis was atypical pneumonia.

*Case 4:* A white housewife, aged 40, was hospitalized three weeks after the onset of symptoms of malignant hypertension. Her temperature was 101 degrees F., pulse 106, respirations 22, and blood pressure 225/137. She had secondary anemia, and a white count of 10,500 to 23,400 with 89 per cent neutrophils. She had no cough nor sputum. Rhonchi were heard throughout the lungs terminally as the woman lapsed into coma. She expired on the 13th hospital day. Necropsy revealed malignant nephrosclerosis, cardiac hypertrophy, acute fibrinous pericarditis, and bronchopneumonia. The right lung weighed 765 and the left, 585 grams. Both lungs were edematous and crepitant, but the lower lobes, especially the right, exhibited consolidations that were grayish yellow, flabby, and vaguely demarcated. Microscopically, the lungs were extensively hyperemic and hemorrhagic. The alveoli contained sparse scatterings of mononuclear cells, occasionally were lined by cuboidal septal cells, and some contained plugs of hyalinized exudate. Lobular and sublobular areas were infiltrated by neutrophils. The histologic diagnosis was atypical pneumonia.

*Case 5:* A white housewife, aged 21, had had bilateral bronchiectasis since childhood. She developed bronchopneumonia soon after childbirth one month previously. Upon hospitalization, she was irrational, cyanotic, and dyspneic. Temperature was 101.4 degrees F., pulse 136, respirations

60, and blood pressure 120/50. Breath sounds were noisy and coarse rales were heard, especially over the left side. The white count was 14,300 with 78 per cent neutrophils. X-ray film inspection showed extensive infiltrative densities in the right lower lobe and left upper two-thirds, and cardiac enlargement. The woman died about five weeks after the onset of her illness. Necropsy revealed extensive bilateral bronchiectasis and bronchopneumonia, serofibrinous pleuritis, and right-ventricular cardiac hypertrophy. The right lung weighed 870 and the left, 805 grams. On section, the moist surfaces were pinkish to bluish red with numerous scattered indistinct yellowish red, flabby consolidations, confluent in the left upper lobe. The pus-filled dilated bronchi were most prominent in the left lower lobe. Microscopically, the consolidated areas contained much fibrin, many erythrocytes, and some neutrophils. In some zones, alveoli and ducts contained plugs of similar exudate, with admixture of lymphocytes, plasma cells, and a few fibroblasts, bearing a similarity to early stages of Masson bodies. Occasional bronchioles showed changes greatly resembling those in Case 2. The histologic diagnoses were bronchiectasis and atypical pneumonia.

*Case 6:* A white coal-miner, aged 39, who had been ill for six months with chronic bronchitis, polyarthritis, and dermatitis herpetiformis, developed fever, myocardial insufficiency, dyspnea, and cyanosis. During the last six weeks, temperature ranged from 98.6 to 102.8 degrees F., pulse from 92 to 144, and respirations from 16 to 44, with blood pressure 95/50. The white count went from 5600 to 7100 with a maximum of 84 per cent neutrophils. A slight cough was nonproductive. X-ray film revealed prominent bilateral hilar and peribronchial lymphatic markings, especially in the right lower lobe. Necropsy revealed upper right fibrous pleural adhesions, generalized cardiac dilatation, and bilateral bronchopneumonia. The edematous and firm right lung weighed 1170, and the relatively similar left, 780 grams. On section, dull red softly-fleshy areas were scattered throughout. Microscopically, the right lung showed a mild chronic pleuritis. The alveoli generally were lined by proliferated septal cells and contained serous exudate with many monocytes, pigmented macrophages, erythrocytes, and neutrophils. Numerous alveoli and alveolar ducts were lined by hyaline membranes or plugged with coagulated fibrin, and some contained vascular fibrous granulomas infiltrated with plasma cells. The bronchioles contained mucus and neutrophils, and their walls were infiltrated by lymphocytes and neutrophils. The histologic diagnosis was bilateral atypical pneumonia.

*Case 7:* A white gardener, aged 63, developed coronary thrombosis with myocardial infarction, complicated by bronchopneumonia. He had a temperature of 97 degrees F., pulse of 98, respirations of 26, and blood pressure of 156/100. Medium and fine rales were heard throughout both lungs, especially the right upper lobe. X-ray film showed pulmonary congestion, particularly on the right, and some cardiac enlargement. Electrocardiogram indicated left-ventricular hypertrophy and old lateral myocardial infarction. The white count was 18,700 with 81 per cent neutrophils. The man had a dry cough and became dyspneic and cyanotic. Temperature reached 102 degrees F. He became comatose and expired in 17 days. Necropsy disclosed severe coronary sclerosis, ruptured left posterior papillary muscle, extensive myocardial infarction and fibrosis, and bilateral bronchopneumonia with fibrinous pleuritis. The right lung was heavier

than normal. The basal two thirds of the upper lobe contained finely granular and nodular, flabby grayish red to yellow areas, as did the left lung in its upper lobe centrally. Microscopically, wide spread inflammatory changes were apparent. The hyperemic, hemorrhagic alveolar walls and the lumina were infiltrated by macrophages. Many alveoli exhibited swelling and proliferation of septal cells and occasional fibrin plugs, some of which were partially organized. Scattered subiobular foci contained neutrophils. Occasional smaller bronchi contained a neutrophilic exudate. The histologic diagnosis was bilateral atypical pneumonia.

*Case 8:* A retired white stonecutter and miner, aged 74, had generalized arteriosclerosis, congestive heart failure, and extensive bilateral bronchopneumonia. His temperature ranged from 98 to 101 degrees F., pulse from 112 to 120, and respirations from 32 to 40. Blood pressure was 94/70. The white count was 4400 with 88 per cent neutrophils. Moist crackling rales were heard, especially on the left basally; breath sounds were coarse. The man was weak, expectorated a little bloody mucoid sputum, and became progressively more dyspneic and cyanotic. X-ray film revealed evidence of infiltrative changes in both hilar regions and along the bronchial trees. Death occurred six days after admission. Necropsy revealed aortic and mitral stenosis, cardiac hypertrophy, pronounced visceral passive hyperemia, and bilateral bronchopneumonia. The right lung weighed 980, and the left, 840 grams. Both were edematous and dark red on section. Microscopically, many alveoli contained fibrin, neutrophils, and pigment-laden macrophages; others were hemorrhagic. Numerous alveoli and ducts were lined by hyaline membranes; others showed prominent septal-cell proliferation. Occasional arterioles and alveolar walls exhibited fibrinoid swelling. Several smaller veins were plugged by thrombi. The histologic diagnosis was bilateral atypical pneumonia.

*Case 9:* A white retired railroad man, aged 66, became ill suddenly, with vomiting, bloody sputum, weakness, cyanosis, and increasing dyspnea. His temperature ranged from 98 to 100° F., and pulse from 96 to 115; respirations were 30 and blood pressure 112/44. The white count was 31,800 with 90 per cent neutrophils. Scattered rales were heard basally throughout both lungs, more extensively on the right. X-ray film displayed cardiac enlargement, dilatation of the aorta, and finely-mottled pulmonary infiltration, chiefly hilar and basal. Cyanosis and dyspnea progressed. Following an attack of projectile vomiting and severe pain in the chest, the man went into shock and expired eight days after the onset of the illness. Necropsy disclosed rupture of the ascending aorta, secondary to medial necrosis, hemopericardium, cardiac dilatation, and bilateral bronchopneumonia. The lungs were not enlarged and had small indistinct consolidated foci in the right upper and middle lobes, and the left lower lobe. Microscopically, the alveoli contained sparse but diffuse exudates composed of monocytes, deposits of fibrin, and hyaline membranes, as well as some pigment-laden or fatty macrophages. Blood vessels were hyperemic. There were occasional subiobular infiltrations by neutrophils. The histologic diagnosis was bilateral atypical pneumonia.

*Case 10:* A white salesman, aged 37, developed pneumonia following coronary thrombosis and myocardial infarction. His temperature was 99 degrees F., pulse 130, respirations 16, and blood pressure 132/86. He was slightly cyanotic. Electrocardiogram indicated an acute anterior left-ventricular infarction. The white count was 18,600 with 86 per cent

neutrophils. A pericardial friction rub was heard. Cough, with a slight amount of rusty mucoid sputum, developed after two or three days, when the temperature rose to 101.6 degrees F., and respirations to 32. Death occurred on the 12th day. Necropsy revealed severe coronary sclerosis, occlusive thrombosis of the anterior descending branch of the left coronary artery, extensive recent infarction of the anterior left-ventricular wall and septum with mural thrombosis and serofibrinous pericarditis, and pneumonia of the left lower lobe. The lungs were edematous; the left lower lobe was extensively consolidated in softly and vaguely nodular patches. Microscopically, this portion exhibited proliferation of septal cells that formed a continuous lining in many groups of alveoli, hyaline membranes, clusters of macrophages, fibrin plugs, and occasional small cellular-fibrous granulomas in alveoli and alveolar ducts. Histological diagnosis was atypical pneumonia of the left lower lobe.

The definite clinical diagnosis of primary atypical pneumonia was not made in any case. Pulmonary symptoms were present in most instances, but cardiovascular disease was the dominating feature in several patients. It is noteworthy that there were three cases of myocardial infarction and one of ruptured aorta. Myelogenous leukemia, postpartum hemorrhage, malignant nephrosclerosis, and bronchiectasis were the main conditions in other patients. The clinical symptomatology including leukocytosis in most cases was invariably too severe to suggest nothing more than "atypical pneumonia," usually a mild nonfatal disease. In most instances, therefore, the atypical pulmonary inflammatory reaction, detected on pathologic examination, supervened upon a pre-existing ailment or debilitating condition.

### *Pathology*

Grossly, the most important pulmonary changes were increase in weight and ill-defined, somewhat nodular consolidations of lobular or smaller dimensions. The lungs weighed from 500 to 1600 grams each and were variously edematous and hemorrhagic. The bronchi and blood vessels were not remarkable.

Microscopically, the changes were more widespread than the gross appearance had suggested. The alveolar walls showed congestion, edema, and fibrinoid swelling. Septal-cell proliferation was prominent, and intraluminal ribbons of exfoliated cells were numerous. The lumina contained macrophages, erythrocytes, granular precipitate, and clumps of fibrin. In some instances, macrophages were laden with fragments of fibrin. Scattered areas revealed prominent hyaline membranes. Occasionally, there were organized plugs of fibrinous exudate that resembled the Masson bodies observed in rheumatic pneumonia.[1] Some of these fibrinous plugs were covered by a membranous layer of septal cells. Infiltration with segmented leukocytes played a minor role; it was

FIGURE 1 FIGURE 2 FIGURE 3

*Figure 1:* Fibrin plugs in alveoli and alveolar walls. Peppering with polymorphonuclear leukocytes. (x175).—*Figure 2:* Fibrinoid swelling of alveolar wall and hyaline membrane. (x230).—*Figure 3:* Fibrinoid swelling of wall of small artery. (x275).

associated occasionally with clumps of bacteria. Neutrophils and necrotic nuclear debris in both lumina and walls produced the effect of peppering. Sometimes, small arteries and veins exhibited fibrinoid swelling and, more rarely, recent thrombosis. Bronchiolitis obliterans and peribronchial infiltration, chiefly lymphocytic, were seen in two cases (Cases 2 and 5). No intracellular inclusion bodies were detected within bronchial or bronchiolar epithelium.

*Comment*

In past studies on pulmonary changes in chicken pox and rheumatic fever, we[2,1] described histologically similar lesions, which are related undoubtedly to those in primary atypical or viral pneumonia. The histologic features in this form of pneumonia have been described by various authors, including McNaught,[3] Golden,[4] Parker, Jolliffe and Finland,[5] and Saphir.[6] A thorough review of the whole problem was given recently by Reimann[7] and of the pathologic aspects by Anderson.[8] Anderson emphasized the following histologic features: edema and predominently mononuclear-cell infiltration of the bronchial and bronchiolar submucosa; desquamation, ulceration, and not infrequently squamous metaplasia of the bronchiolar lining epithelium; cellular and fluid exudate radiating into peribronchial tissue and alveolar walls, with great thickening of the walls; serous and mononuclear-cell exudate in the alveolar spaces; hyaline membrane; presence of alveolar lining cells.

Although our series conforms clinically not at all or only in part to the descriptions of primary atypical pneumonia, we feel that the pulmonary inflammatory reactions are "atypical" in comparison with bacterial lesions of the lungs, that they belong actually in the group of atypical pneumonias, and that a virus alone or in combination with other agents plays an important etiologic role.

Some of the features in our series are uncommon in primary atypical pneumonia, namely, fibrinous plugs, Masson bodies, "fibrinophagia," fibrinoid swelling of the walls of blood vessels, and bronchiolitis obliterans. The histogenesis of the Masson bodies, their relation to fibrinous plugs, and their differentiation from the fibrous changes in chronic pneumonia, as well as the significance of the vascular changes, were discussed in our paper on rheumatic pneumonia[1] and also more recently by Mossberger.[9] The presence of alveolar macrophages tending to fragment and phagocytose fibrin—"fibrinophages"—was a peculiar feature that we had not observed previously. The occurrence of bronchiolitis obliterans was not surprising inasmuch as organization of plugs

FIGURE 4 FIGURE 5 FIGURE 6

*Figure 4:* Disintegration of fibrin plugs with "fibrinophagia," and mononuclear-cell exudate. (x200).—*Figure 5:* Septal-cell proliferation forming a cuboidal-cell lining in alveoli containing exudate. (x175).—*Figure 6:* Masson bodies and mononuclear-cell exudate. (x115).

of exudate in bronchioles with ulcerated mucosae has been described by Hart and Mayer[10] in related forms of pneumonia such as that of influenza and measles. Hyaline membranes, a common finding in primary atypical pneumonia, were seen repeatedly in our material. We feel that the eosinophilic homogeneous hyaline membranes resulted from the coalescence of the noncellular exudate pushed, under increased intra-alveolar pressure, against the alveolar and ductal walls that had undergone fibrinoid swelling and necrosis. The significance of "Quellungsnekrosen" in the alveolar walls was discussed recently by Kuehn and Pichotka.[11] Interstitial infiltrations were less conspicuous in our material than in classical cases of primary atypical pneumonia. The glandlike proliferation of alveolar lining cells resembled the picture seen in chronic progressive pneumonia in sheep and in human pulmonary adenomatosis.

Atypical pulmonary reactions as described in this paper appear to be much more common than formerly believed. They may supervene in a variety of diseases and cause fatal termination in persons without clinical evidence of pneumonia. Their occurrence in individuals having myocardial infarction must be more than coincidental. If the lesions are of viral etiology, the virus or vira must be common and capable of being pathogenic in persons who have been debilitated by other diseases, especially diseases of the heart. Apropos of this idea, the recent work of Pearce and Lange[12] is of interest. They found that in rabbits the incidence and severity of viral lesions of the heart were increased greatly when the animals were submitted to procedures that tended to decrease the amount of oxygen supplied to the heart. One may conjecture that inadequate oxygenation facilitates viral action in the human lung.

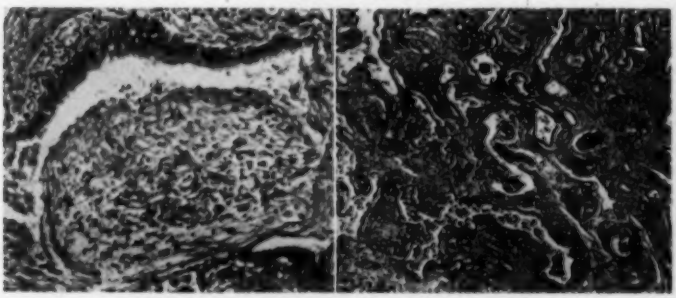

FIGURE 7 FIGURE 8

*Figure 7:* Bronchiolitis obliterans. (x175).
*Figure 8:* Interstitial fibrosis with adenomatoid septal-cell proliferation. (x90).

The relatively large number of individuals with cardiac failure in this series raised the question as to the difference between atypical pulmonary reactions and passive congestion of the lungs. Therefore, a study was made of a control group of 10 cases of acute and subacute passive pulmonary congestion secondary to cardiac failure that was due chiefly to coronary occlusion and myocardial infarction. In these instances, the pulmonary lesions were more diffuse. The alveoli showed varying degrees of congestion and edema of the walls, and accumulation of pigmented phagocytes in the lumina. Septal-cell swelling and proliferation were far less prominent. Hyaline membranes and foci of organized exudate or granulomas were absent. Thus, the histologic changes in uncomplicated acute and subacute passive congestion of the lungs were different from the atypical pulmonary inflammatory reactions observed in this series.

The similarity of the histologic features described here to the lesions of primary atypical pneumonia and of known viral pneumonias, as well as of rheumatic pneumonia, is so striking that the question of viral etiology should be considered in all these conditions. In our paper on rheumatic pneumonia,[1] we mentioned that the pattern of the lesions was suggestive of viral etiology—is it possible that a nonspecific virus produces in rheumatic patients what is called "rheumatic" pneumonia? Or are we dealing with an anaphylactic reaction, as believed by Rich and Gregory,[13] Mossberger,[9] and others?

We still believe that the histologic pattern of "rheumatic pneumonia" when found in a rheumatic patient is characteristic. Other recent writers (Mossberger,[9] Kraevski,[14] Muirhead and Haley,[15] Seldin, Kaplan and Bunting,[16] and Maksimovich[17]) expressed the same opinion. However, we should be reluctant today to call the picture "specific." Herbut and Manges[18] had already contested the specificity of the Masson body; they found these granulomas "in a high percentage of cases of organizing pneumonia, pulmonary abscess, bronchiectasis, and tuberculosis." Their findings were in contrast to our former observations on control material in which Masson bodies occurred only exceptionally and, in particular, not as a feature of chronic organizing pneumonia. Nevertheless, in the more recent years, we have seen several cases, in addition to the series reported in this paper, which presented a similar histologic pattern but gave no evidence of rheumatic disease. Therefore, we feel that the Masson body should not be considered as specific a granuloma as the Aschoff body.

In closing, we should like to mention the possible sequelae of "atypical pulmonary inflammatory reactions." Interstitial fibrosis may develop from interstitial inflammation, and minute fibrous

patches may result from involutional changes in Masson bodies. Adenomatoid structures may be traced back to proliferated septal cells and, possibly, to bronchial epithelium. In other words, such new disease entities as "diffuse progressive interstitial fibrosis" of the lungs and "pulmonary adenomatosis," perhaps even alveolar-cell tumor, may be considered late sequelae of atypical pneumonic lesions. Anderson,[8] Ferrari et al.,[19] and Mallory[20] have entertained similar trends of thought.

The publications on interstitial fibrosis of the lungs (Hamman and Rich;[21] Eder, Hawn and Thorn;[22] Potter and Gerber;[23] Beams and Harmos;[24] Meessen[25]) show the variations in the duration of this condition; some of its histologic features resemble those seen in "atypical pulmonary inflammatory reactions," and its development from atypical pneumonia is at least a possibility. We believe that progressive interstitial pulmonary fibrosis is not too rare; figure 8 shows its histologic pattern in a woman, aged 80, in whom the lesions were limited to the patchily-consolidated, darkly grayish red lower lobe of the left lung, which weighed 430 grams. The woman died a few hours after hospitalization, and no reliable data on the previous history were available; however, the picture is very suggestive of being a late stage in the development of "atypical pulmonary inflammatory reaction." It greatly resembles photographs in other pertinent papers, and also figure 16 in the paper of Waddell, Sniffen and Sweet,[26] illustrating a late stage of chronic pneumonitis with cholesterol deposits, which were not seen in our material. The relation of interstitial fibrosis to lesions of pulmonary adenomatosis is likewise apparent in the photographs; the epithelioid lining of distorted alveoli and ducts produces an adenomatoid pattern. Pulmonary adenomatosis may develop after an acute respiratory infection (Bubis and Erwin[27]). Although no specific etiologic factor has been established in this condition (Swan[28]), its viral etiology is being considered more and more (Stephens and Shipman[29]).

## SUMMARY

1) A series of 10 cases has been presented with atypical pulmonary inflammatory reactions, probably of viral origin, that resemble closely those seen in chicken pox, rheumatic fever, and in primary atypical pneumonia.

2) The pulmonary changes failed to produce a clearcut clinical syndrome. As a rule, they supervened upon other diseases, predominantly cardiovascular disturbances.

3) The lesions include fibrinoid swelling of the walls of alveoli and blood vessels, septal-cell proliferation, mononuclear-cell exudate, hyaline membranes, occasional peppering with neutrophils,

fibrinous plugs, "fibrinophagia," Masson bodies, and bronchiolitis obliterans. Inclusion bodies were not seen.

4) The inclusion of these reactions, thus described, among the larger group of atypical pneumonias of diverse etiology appears justified on the ground of their histologic pattern.

5) Presence of Masson bodies should no longer be considered as occurring only in rheumatic pneumonia.

6) Diffuse progressive interstitial pulmonary fibrosis, pulmonary adenomatosis, and alveolar-cell tumor of the lung may result from such atypical pulmonary inflammatory reactions.

*Acknowledgment:* We are indebted to Dr. J. Z. Appel and to Dr. T. G. Boughton for the privilege of inclusion of Case 2 in this study.

## RESUMEN

1) Se presenta una serie de 10 casos con reacciones pulmonares inflamatorias atípicas, probablemente causadas por virus, que se asemejan bastante a las de la varicela, la fiebre reumática y la neumonía primaria atípica.

2) Las alteraciones pulmonares no produjeron síndromes clínicos bien definidos. Por lo general, fueron complicaciones de otras enfermedades, especialmente de las afecciones cardiovasculares.

3) Las lesiones incluyen tumefacción fibrinosa de las paredes de los alvéolos y de los vasos sanguíneos, proliferación de las células de los septums, exudados de células mononucleares, membranas hialinas, agrupaciones de neutrófilos en ocasiones, tapones fibrinosos, "fibrinofagia," cuerpos de Masson y bronquiolitis obliterante. No se observaron cuerpos de inclusión.

4) Parece justificarse la inclusión de estas reacciones entre el grupo más grande de las neumonías atípicas de etiología diversa, cuando se considera el cuadro histológico.

5) Ya no se debe considerar que los cuerpos de Masson sólo pueden ocurrir en la neumonía reumática.

6) Estas reacciones pulmonares inflamatorias atípicas pueden causar fibrosis pulmonar intersticial difusa y progresiva, adenomatosis pulmonar y tumores del pulmón de células alveolares.

## RESUME

1) Les auteurs rapportent 10 observations de réactions inflammatoires atypiques du poumon vra isemblablement dues à un virus. Ces cas ressemblent de très près à ceux que l'on peut voir au cours de la varicelle, de la fièvre rhumatismale, et dans les pneumonies atypiques primaires.

2) Les altérations pulmonaires ne sont pas capables de constituer un syndrome nettement défini. En règle, elles ne sont qu'un

épiphénomène au cours d'autres manifestations et en particulier cardio-vasculaires.

3) Les lésions comprennent une tuméfaction fibrinoïde des parois des alvéoles et des vaisseaux, une prolifération des cellules interstitielles, un épanchement d'éléments mononucléaires, des membranes hyalines, dans certains cas des amas fibrineux parsemés d'éléments neutrophiles, des corpuscules de Masson, et une inflammation oblitérantes des bronchioles. Il n'a pas été mis en évidence de corps d'inclusion.

4) L'homologation des réactions que nous venons de décrire dans le groupe plus important des pneumonies atypiques d'étiologie diverse se justifie par leurs caractères histologiques.

5) La présence de corpuscules de Masson ne doit plus être considérée comme propre à la pneumonie rhumatismale.

6) Certaines scléroses pulmonaires interstitielles et certaines tumeurs des cellules alvéolaires peuvent être la conséquence de ces réactions inflammatoires de pneumonie atypique.

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# Spontaneous Hemopneumothorax: Treatment by Early Thoracentesis*

## Report of Four Cases

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Spontaneous hemopneumothorax as a clinical entity is being recognized with more and more frequency. Early recognition and treatment is so important in avoiding unnecessary morbidity and mortality that this condition must be considered in the differential diagnosis of all episodes of acute chest pain. Thus far approximately 58 cases have been reported in the literature, with reviews on the subject being written in 1942 by Hartzell[1] and in 1949 by Hansen.[2]

During the past two years at the Kings County Hospital, four cases of spontaneous hemopneumothorax were observed. One case occurred in a patient with congenital cystic disease of the lung, making this case the third to be reported in the literature.[2] The other three were in young males ranging in age from 17 to 35 years with no etiology being found either at the time of the acute episode or on nine months to two years follow ups. Two of the cases studied occurred in males under 20 years of age, an age group rarely affected by this condition.[1] A case with fatal termination was also seen in a 58 year old man but necropsy revealed the presence of multiple tuberculous abscesses in both lungs. This case is not being included in our report. All cases were observed in males, a finding consistent with that of other authors, only three cases having been reported thus far in women.[2] The reason for this sex incidence is not known.

It is generally agreed that this condition usually occurs in healthy individuals and results from the rupture of an emphysematous bleb and subsequent tearing of small vessels which are present in adhesions between the pleura and chest wall.[3] It is felt that the rupture of the bleb occurs first and as air enters the pleural cavity the lung is drawn away from the chest wall and tearing of adhesions and blood vessels within these adhesions takes place. It is interesting to note that in two of the following cases there was a history of previous pneumonic infection which could well have accounted for the presence of adhesions.

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The onset of spontaneous hemopneumothorax is not usually precipitated by trauma or strain, and is in no way related to active tuberculosis or other pulmonary infections. Although large amounts of blood and air in the pleural cavity may rarely occur during the course of active tuberculosis its presence is usually associated with operative procedures or the demonstration of large cavities. Hemopneumothorax occurring during the course of active tuberculosis should not be classified as spontaneous.

The bloody fluid removed in this condition has always failed to clot.[1,4,5] An explanation for the lack of clotting has recently been advanced by Cosgriff[4] who found absence of prothrombin, fibrinogen, and thrombin as well as absence of anti-coagulant activity in the pleural fluid, and concluded that clotting had taken place during the first few hours following the hemorrhage. Apparently the fibrin had been deposited on the surface of the lung and the remaining fluid was now devoid of necessary clotting elements. He felt that at least a portion of the fibrin was later dissolved by a fibrinolytic substance present on the pleural surface. In the following cases the bloody fluid removed from the pleural cavity failed to clot.

The sudden onset of dull or sharp chest paint, followed by increasing dyspnea, perspiration, and weakness is the usual history given by these patients. In some cases abdominal pain is the presenting symptom, and unnecessary laparotomy has occasionally been done.[6] Signs of shock and anemia may be prominent and the patient may appear moribund when first seen. A low grade fever may be present but temperature will usually return to normal upon removal of the hemorrhagic fluid.

The prognosis of spontaneous hemopneumothorax is excellent provided the patient survives the acute episode.

The following cases illustrate some of the points stressed above:

*Case 1:* L.F., a 17 year old white male clerk was admitted to the hospital because of continuous dull chest pain over the right side and increasing weakness of 36 hours duration. Pain had occurred suddenly while he was seated quietly. Two months prior to admission he had had right middle lobe pneumonia which was treated successfully with penicillin. One week before admission a chest x-ray film taken by the board of health showed no abnormality. Physical examination revealed an acutely ill, pale, asthenic male. Pulse 120, respiration 28, blood pressure 110/60, temperature 100 degrees F. A mediastinal shift to the left was noted as well as signs of atelectasis of the right lung and fluid and air in the right pleural cavity. This was confirmed by x-ray and a thoracentesis done on the night of admission yielded 180 cc. of blood. A diagnosis of spontaneous hemopneumothorax was made. Hemoblobin was 11 grams, white blood count was 17,800 with 85 per cent neutrophils. Urine was negative, blood urea was 35 mgm. per cent. Mantoux in 1:100 dilution was negative, and smears and cultures for acid-fast bacilli were negative

on the chest fluid, sputa, and gastric washings. Bleeding, clotting, and clot retraction times were all within normal limits. Thirty-six hours after admission another thoracentesis yielded 800 cc. of blood which had a hemoglobin of 10 grams and a urea of 30 mgm. per cent and which did not clot upon standing at room temperature for two hours. Five days later 1000 cc. of blood was removed. One week after admission a chest x-ray film revealed minimal fluid at the right costo-phrenic sinus and almost full expansion of the entire right lung. Temperature which had been 100 degrees F. returned to normal on the third hospital day. Two weeks later chest x-ray film was completely normal. The patient remained well and nine months after the above episode had a normal chest x-ray film with full expansion of the previously collapsed lung.

*Case S:* B.B., a 35 year old male entered the hospital because of pain in the left chest. At the age of three years he had bilateral bronchopneumonia but had always been in excellent health since then, until 12 hours before admission. While eating dinner he suddenly developed severe tearing pain in the left lateral chest which was followed by dyspnea. No fever, cough or chills were noted. Physical examination revealed an acutely ill male with blood pressure 100/70, pulse 120, temperature 101 degrees F. and respirations 26. Mediastinal shift to the right was present with hyper-resonance above the seventh rib and dullness below over the left chest posteriorly. Chest x-ray inspection confirmed the diagnosis of hydropneumothorax with 85 per cent collapse of the left lung. Five hundred cc. of blood was withdrawn the night of admission. Admission blood urea was 50 mgm. per cent, sugar 88 mgm. per cent. Hemoglobin was 9.5 grams, white blood count 16,600 with 78 neutrophils. Repeated sputa, gastric and pleural fluid examinations were negative for acid fast bacilli. Bleeding, clotting, clot retraction and prothrombin times were normal. Eight days after admission 700 cc. of blood, which did not clot, was removed and x-ray film inspection one day later showed almost total reexpansion of the entire lung and minimal blurring of the costo-phrenic sinus. Immediately after the second thoracentesis the temperature fell to normal and he showed marked clinical improvement. On the 12th hospital day, chest x-ray inspection revealed full expansion of the lung. Two weeks after admission, he was discharged and repeat x-ray films six months, one year, and 18 months later showed no change.

*Case 3:* E.K., a 22 year old white metal polisher was admitted because of left chest pain. He was in excellent health until 20 hours prior to admission, when, while walking home from work, he suddenly noted the onset of severe left chest pain and dyspnea. He went to bed, but because shortness of breath continued, he sought admission to the hospital. No other symptom was noted. Physical examination revealed an acutely ill male with a blood pressure of 98/70, pulse 160, respiration 40, temperature 101 degrees F. Mediastinum was shifted to the right with signs of fluid and air in the left chest. Hemoblobin was 12.5 grams, white blood count 34,600 with 85 per cent neutophils. X-ray inspection showed complete collapse of the left lung with hydropneumothorax. Thoracentesis was done immediately with removal of 350 cc. of blood which failed to clot. Twenty-four hours after admission, another 800 cc. of blood was removed from the left chest. Blood pressure rose to 130/80 and remained at that level. Hemoglobin in the peripheral blood was 10.5 grams; on the chest

fluid, it was 9 grams. Daily chest taps done on the following five days, removed a total of 1500 cc. of blood. He required two transfusions of 500 cc. each and on the 10th day of hospitalization, his hemoglobin was 11 grams. Chest x-ray film on the ninth hospital day revealed complete expansion of the left lung with some blunting of the costo-phrenic sinus. Temperature, which had remained 100 to 101 degrees F., fell to normal on the fifth hospital day and remained so. X-ray films six months, one and two years later, showed no abnormality and the patient has remained in good health.

*Case 4:* S.B., an 18 year old white male was admitted to the hospital because of severe left chest pain and dyspnea of six hours duration. He had a long history of pulmonary difficulties dating back to the age of six years when he had a bout of blood spitting and was told he had pneumonia. Following this, the patient had numerous bouts of hemoptysis and "pneumonia." Acid fast bacilli were never recovered. He continued to experience periodic episodes of hemoptysis but was otherwise in good health until six hours prior to admission when he developed severe left chest pain and shortness of breath. Family history was negative for pulmonary disease. Physical examination revealed an acutely ill white male with blood pressure of 115/80, pulse 100, respiration 28, temperature 98 degrees F. He splinted his left chest and signs of a hydropneumothorax were present on that side. X-ray inspection confirmed the above and in addition, revealed the presence of numerous areas of cyst formation within the lung parenchyma (Fig. 1). Admission hemoglobin was 11.5 grams, hematocrit was 43 per cent, white blood count was 13,200 with a normal differential. Blood urea was 22 mgm. per cent, sugar was 106 mgm. per cent, total protein was 7.5 mgm. per cent. E.K.G. showed changes compatible with pulmonary hypertension. Thoracentesis done

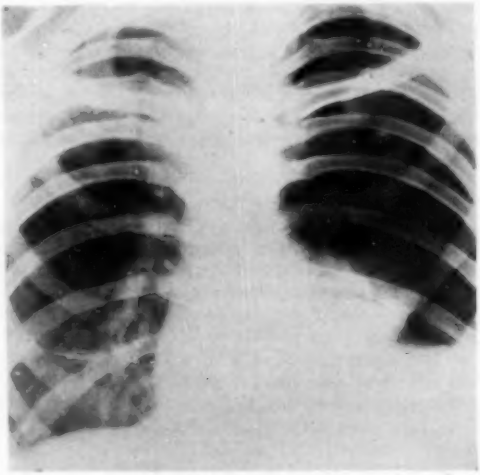

FIGURE 1: Hydropneumothorax with collapse of the left lung.

on the night of admission yielded 650 cc. of blood with a hemoglobin of 13.5 grams, hematocrit of 41 per cent, specific gravity of 1050 and a total protein of 6.75 grams. One day later 350 cc. of blood was removed and subsequent thoracenteses on the third and fourth hospital days removed 200 and 300 cc. of blood. Fourteen days after admission chest x-ray inspection revealed complete reexpansion of the left lung. In addition, two large areas of radiolucency measuring 8 x 10 cm. with calcification of the walls were noted in the right mid-lung field (Fig. 2). The picture was felt to be consistent with a diagnosis of cystic disease of the lung. He was discharged on the 20th hospital day and remained asymptomatic for the next two and one half years. X-ray film taken 25 months after the above episode showed no change when compared with previous films.

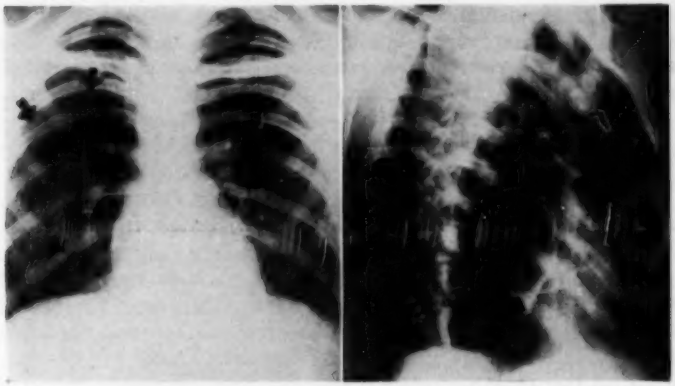

FIGURE 2a FIGURE 2b

*Figure 2a:* Fourteen days after the acute episode. Complete reexpansion of the lung has occurred. A large calcified cyst is seen in the right lung field.—*Figure 2b:* The lung cyst is well outlined in the right oblique projection.

*Comment:* The treatment of spontaneous hemopneumothorax has long been a subject for debate. Head[7] and Hopkins[6] feel that the fluid should be removed cautiously and replaced by air so that intrapleural positive pressure remains high. These observers state that the creation of negative intrapleural pressure by the withdrawal of fluid and air might cause the hemorrhage to continue or recur. Head also feels that transfusions should be given cautiously and that blood pressure should not be raised too rapidly for fear of forcing out the clot in the bleeding vessel. More recently Dorset and Terry[8] have recommended removal of small amounts of fluid over a relatively long period of time.

The experience of Simpson[9] and Johnson[10] however, does not appear to bear this out. These authors stress the fact that there is no danger in rapid removal of the hemorrhagic fluid by early and repeated thoracenteses. On the contrary, it is felt that this

is the treatment of choice and that morbidity and operative intervention can be kept at a minimum by this method. Johnson treated 350 cases of traumatic hemopneumothorax in this way with excellent results. Transfusions should be given to correct the anemia and other measures that are routinely employed to combat shock should be used. Rapid evacuation of the fluid and air is important not only as an emergency measure to relieve symptoms and effects of mediastinal shift, but is of value in preventing further fibrin deposition and constrictive pleurisy. If excessive amounts of fibrin and dense adhesions are allowed to form, surgical intervention and decortication is often necessary to obtain reexpansion of the lung. Spontaneous absorbtion of the hemorrhagic fluid may occur but the process usually is a slow one and residual constrictive effects are more apt to result if this is allowed to take place.[10] Although infection of the fluid is rare in this condition, empyema and subsequent lung retraction have been reported.[3] This complication can also be prevented by early aspiration.

The four cases of spontaneous hemopneumothorax presented above were all treated by prompt and repeated thoracenteses. All of the patients improved considerably following the removal of fluid and air, and respiratory and circulatory embarrassment were almost immediately corrected by this procedure. Repeated or increasing hemorrhage did not occur following chest taps and in only one case were transfusions necessary (case 3). The patients all recovered completely and full lung expansion occurred within seven to 13 days after the onset of the acute episode. In the cases reported by Hartzell,[1] Hansen,[2] and Dorset and Terry,[5] where more conservative management was practiced, full expansion of the lung did not occur until 14 to 40 days following hospitalization, and presence of pleural fluid or pleural thickening was often demonstrable by x-ray inspection many months after onset of the illness. It would appear from these findings that the morbidity and length of hospital stay were markedly reduced by treatment of these cases with early and repeated chest taps. The first three patients studied were seen at intervals for periods of one to two years and were all well and had essentially normal chest x-rays when last observed. The patient with congenital cystic disease of the lung (case 4) had remained in good health with no recurrence of hemoptysis, but the lung cysts were still visible on x-ray inspection.

## SUMMARY

Four cases of spontaneous hemopneumothorax are reported occurring in males from 17 to 35 years of age. All cases were treated by early and repeated thoracenteses with rapid and complete recovery.

## RESUMEN

Se comunican cuatro casos de hemoneumotórax espontáneo que occurieron en hombres de 17 a 35 años de edad. En todos los casos se emplearon toracentesis tempranas y repetidas y todos recuperaron rápida y completamente.

## RESUME

L'auteur rapporte 4 cas d'hémopneumothorax spontanés survenus chez des individus de 17 à 35 ans. Tous ces cas furent traités par des thoracentèses précoces, et répétées, suivies de guérison rapide et complète.


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# Bronchoscopy in Tuberculosis

## Deformity of the Major Bronchi Associated with High Diaphragm Rise in Certain Forms of Non-Surgical Collapse Therapy

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The primary object of this paper is to present some observations on the configuration of the bronchial tree: 1) in the normal, and 2) following collapse therapy, and from these observations to point out some limitations of collapse therapy.

Before discussing the subject at hand, it should be borne in mind that there are deficiencies in our present knowledge of pulmonary physiology and in the therapy of tuberculosis. Therapy is still more or less a trial-and-error affair, recent advances in drug treatment notwithstanding. Bed rest and collapse measures remain the mainstays and the underlying principles are largely mechanistic. The factors involved are many and their relative worth equivocal; therefore, the pitfalls resulting from hasty or erroneous conclusions warn against venturing too far beyond the bounds of known facts. To make an observation of fact is one thing; to judge the true and relative value of that observation is another. An open mind and willingness to let the light of time decide the merit of the case are essential.

The usual textbook description of the main bronchi states that the right main bronchus makes an angle of 25 degrees with the midline and the left main bronchus makes a 75 degree angle, presumably in a straight line. Figure 1 shows the bronchogram of a young policeman made during a fruitless search for the source of an isolated hemoptysis. In this illustration the relatively straight course of the right main bronchus is apparent. On the left side is shown the angle with the midline, but more significantly it can be noted that the axis is not a straight line nor does the bronchus lie in one plane throughout its course. There is present an inferior convexity in the shape of a shallow "U". One arm of this "U" is at the carina and the other continues into the upper lobe bronchus. Gently sloping from the under side of the "U" is

*From the Department of Medicine and Surgery, Veterans Administration, Bay Pines, Florida. (Based on work at Veterans Administration, Oteen, North Carolina).

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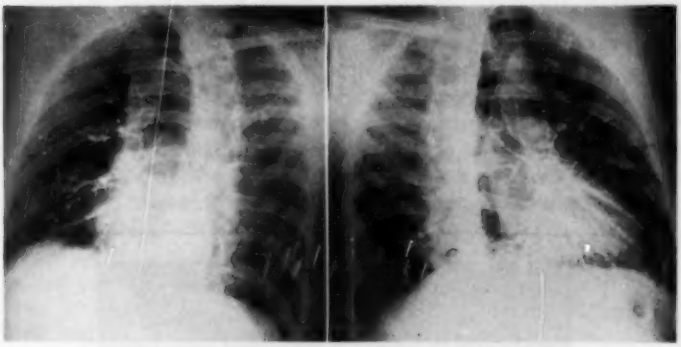
the lower lobe bronchus. Note the mild dorsal curvature of the lower lobe bronchus.

A diaphragm rise on the right induced by either phreniclasia and/or a pneumoperitoneum is directed perpendicular to the long axis of the main bronchus. Only in the more marked rise (to the second or third anterior interspace) does an appreciable deformity occur. The most constant deformity seen on the right side is a buckling at the level of the middle lobe orifice; the bronchus turns sharply laterally and then after a distance of approximately one centimeter resumes a semi-vertical course to the lower lobe open-



Right Tree Left Tree

FIGURE 1: Normal Bronchi—Oblique views.

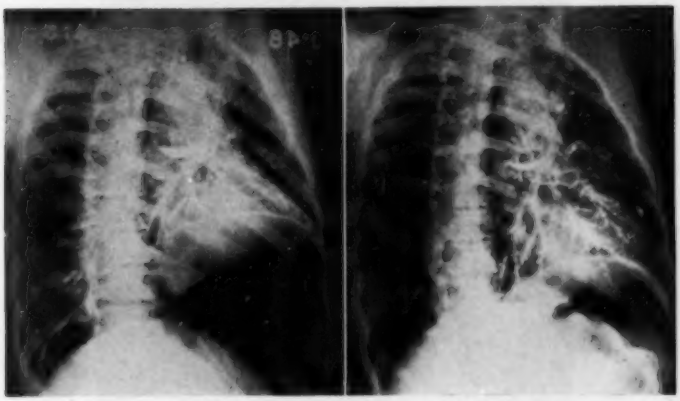


FIGURE 2a FIGURE 2b

*Figure 2a:* Left Tree (February 20, 1948).
*Figure 2b:* Left Tree (March 17, 1948).

ings. Concomitantly there is a variable degree of foreshortening and symmetrical narrowing of the lumen. Interference with drainage is not apt to be significant.

On the left side, however, the picture is entirely different. With a marked diaphragm rise a lifting action is exerted on the convex underside of the "U" described above for the normal, and the 75 degree angle approaches 90 degrees. This is illustrated in Figures 2 and 3. The dorsal curvature of the lower lobe bronchus becomes a definite angulation which starts above the level of the upper lobe orifice incorporating a portion of the main bronchus. The bent distal portion approaches a perpendicular relation to the "U" curve. There is also generalized foreshortening and lateral compression. The lower lobe openings, the nest-of-three basal branches, are compressed, sometimes to one-half or less of their usual area. At times only one lower lobe septum can be visualized through the narrow lower lobe bronchus. The upper lobe orifice may be reduced to a superior-inferior slit. The picture as viewed through the bronchoscope is as follows:

There is marked lateral deviation of the main bronchus. A

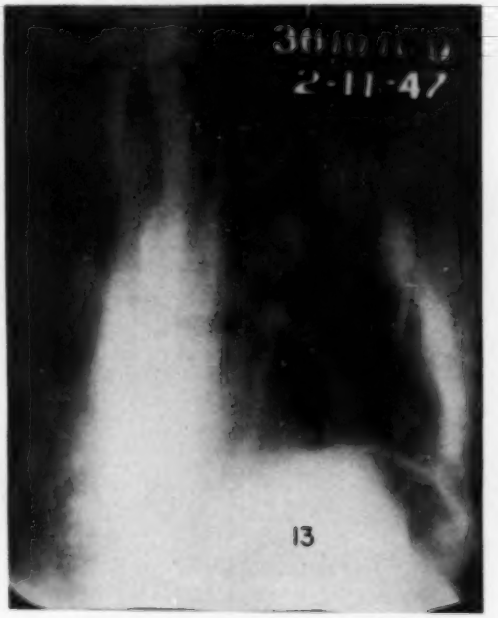



FIGURE 3: Laminograph—Left Main Bronchus.

similar effect occurs when the trachea is deflected to the right, as from right upper lobe atelectasis. As the region of the dorsal angulation or drop-off is approached, the scope may be so bent that only a portion of the distal opening can be kept in view. As a rule, only the extreme upper portion of the upper lobe spur can be seen hidden beyond the angulation. Often only after the scope has been passed into the lower lobe bronchus can the upper lobe orifice be seen; in the process the bronchus is straightened on the scope and the latter is then slightly withdrawn to the upper lobe orifice level.

Figure 2 illustrates within the limitations of a bronchogram what happens when the diaphragm level is changed on the left side by less than one interspace, even though a relatively high level is maintained before and after. The deformity pattern is well shown in Figure 2a. One month later the amount of air beneath the diaphragm was reduced; the diaphragm dropped about three-fourths of an anterior interspace, and a bronchogram was again made (Figure 2b). The lower lobe bronchus can be seen to lengthen and widen and the secondary branches to open in all directions. While these findings can be appreciated, to some extent, by bronchography and laminography, they are most impressive when seen through the bronchoscope (Figure 4). In other words, at high diaphragm levels a small rise may be critical.

The manner in which this rather constant deformity was called to the author's attention is interesting and significant. When streptomycin was first introduced in 1946 for the treatment of endobronchial tuberculosis, those cases with non-stenosed, ulcerogranulomatous lesions which were *most resistant* to previous therapy were selected for trial. Of nine cases selected, seven were of left-sided lesions and in six of these there was marked deformity of the bronchial pattern as described above, associated with a high diaphragm level induced by pneumoperitoneum and left phreniclasia.

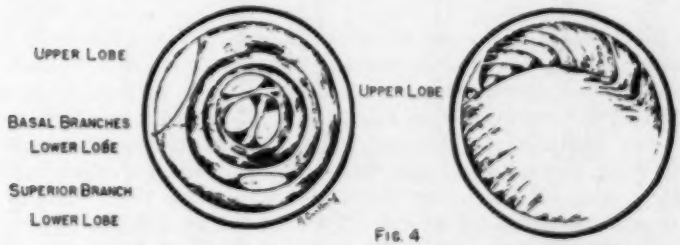



FIG. 4

B.L.B., WHITE MALE, AGE 22 YEARS

| Date | Collapse Therapy | Bronchoscopy | Diaphragm Level | Remarks |
|---|---|---|---|---|
| 5/22/47 | | | 5 A. IS | No collapse therapy. Cavitation, LUL. |
| 6/ 2/47 | Phreniclasia, left. | | | |
| 6/ 6/47 | Pneumoperitoneum started. | | | |
| 6/ 9/47 | | | 4 A. Rib | |
| 8/22/47 | | | 3 A. Rib | |
| 12/ 1/47 | | | 3 A. Rib | |
| 1/29/48 | | Initial bronchoscopy. Reddened mucosa LMB. LULO not seen. | | |
| 2/16/48 | Phreniclasia, left, secondary. | | | |
| 2/24/48 | | LUL spur edematous and thickened. Compression LLL bronchus. Only one lower lobe septum could be seen. | | |
| 3/ 7/48 | | | | Began coughing up ½ cup of dark foul sputum with difficulty. |
| 3/25/48 | (On advice of endoscopist) Pneumoperitoneum reduced. | Increased reddening LMB. LUL spur thicker. | | |
| 4/ 8/48 | | Distortion remains severe with inflammatory reaction unchanged. | | |
| 4/12/48 | Pneumoperitoneum abandoned. | | | On recommendation of endoscopist. |
| 4/22/48 | | | | Gradual cessation foul sputum. |
| 4/26/48 | | Bronchogram shows incipient bronchectatic saccular changes LUL. | | |
| 6/28/48 | | | 4 A. Rib | Cavity smaller. |
| 7/ 9/48 | | | | Cavity closed. |
| 7/13/48 | | Distortion without inflammatory reaction. | 4 A. Rib | Cavity closed. |
| 8/ 4/49 | | | 4 A. Rib | Cavity closed. |

ABBREVIATIONS: A. = Anterior. IS = Interspace. LMB = Left Main Bronchus. LLL = Left Lower Lobe. LUL = Left Upper Lobe. LULO = Left Upper Lobe Orifice.

What is the significance of the observations recorded above? Jackson[1] has pointed out the pathologically important peculiarity of tracheo-bronchial obstructions in that, though small, they set up diffuse destructive secondary changes in the distal portion of the lung out of all proportion to their size. Certainly there is an element of obstruction given the degree of distortion described. Banyai[2] in his work entitled *Pneumoperitoneum Treatment* makes the following statement: "In addition to carefully observing the patient's general condition, it is imperative to ascertain that no greater pulmonary relaxation is produced than is necessary for free bronchial drainage. If pulmonary relaxation oversteps this point, there is a potential danger of causing a kink in the draining bronchi and stopping the drainage. This, of course, is just the opposite of what pneumoperitoneum may accomplish, and therefore must be carefully avoided."

The higher incidence of non-tuberculous bronchiectasis in the left lower lobe is generally ascribed to mechanical reasons. (See description of the normal left tree above). That part of disease-induced secretions over and above that handled by the cilia and body position changes depends upon the cough mechanism for expulsion. With a straight canal, interference with the tussive effort is minimal; with a zig-zag canal, intermittently narrowed, interference is likely. The obstructing nature of the deformity described above, particularly if complicated by endobronchial disease, may be instrumental in converting what may have been a sub-clinical or dry bronchiectasis into a frank clinical entity demanding active therapy.

Furthermore, there is evidence that the deformity of the left tree favors the development of endobronchial disease. A consecutive series of 463 patients bronchoscoped in a tuberculosis hospital was studied (before streptomycin was widely used). Of these, 107, or 23 per cent, were found to have ulcero-granulomatous lesions. Fifty-nine patients had an appreciable deformity of the left tree and in this group the incidence of endobronchial disease was 32 per cent.

Phthisiologists are familiar with the occasional enlargement of a cavity (tension cavity) or an atelectasis with a slight diaphragm rise induced by a phreniclasia or a pneumoperitoneum. This is generally ascribed to the kinking-off of a bronchus draining the area in question. In view of the evidence of deformity of even the major bronchi and their immediate divisions with high diaphragm levels and the marked effect with slight changes in diaphragm height at these levels, it would not seem unreasonable to suggest that optimal levels, depending upon the *individual patient's* response, rather than maximal levels for all cases, be

the goal. In other words, rather than to strive for a maximum diaphragm rise by instituting phreniclasia on the side of maximum disease accompanied by pneumoperitoneum, it would seem wiser to institute pneumoperitoneum in graduated stages. Pneumoperitoneum is a reversible procedure and can be abandoned at will. The level finally obtained (with or without a phreniclasia added at some later date) is then a matter of clinical judgment aided by bronchoscopic observation of the individual bronchial tree for degree of deformity and presence or absence of endobronchial disease. Even though unsatisfactory clinical results in the form of poor drainage of secretions, enlargement of cavity, and an appreciable amount of atelectasis occur only occasionally in this form of collapse therapy, the cautious approach recommended warrants consideration in order that even the occasional case may be salvaged.

An illustrative case which exemplifies most of the points discussed and indicates the role of the bronchoscopist is presented in the form of the accompanying chronological chart.

### *Comment*

In the presence of a markedly deformed left main bronchus, the danger of an engrafted endobronchial disease is augmented and its danger should not be underestimated. A diaphragm rise to a maximal level is not an end in itself. In one patient it may mean pulmonary relaxation and splinting without interference with drainage; in another, however, it may favor the development of the complications discussed, namely, endobronchial disease, enlargement of cavity, bronchiectasis, and atelectasis. Concurrent bronchoscopic study is the only objective means of judging the risk involved in the individual case.

## SUMMARY

1) Collapse therapy to produce a diaphragm rise causes characteristic deformity of the main bronchi. Sharp dorsal angulation of the left main bronchus above the level of all lobe orifices on this side is especially significant.

2) The deformed left tree appears to favor the development of endobronchial disease.

3) Optimal diaphragm rise, rather than maximal rise, based on bronchoscopic observation of the individual case seems the more intelligent approach.

## RESUMEN

1) Las medidas de colapsoterapia que producen elevación del diafragma causan una deformidad característica de los bronquios

principales. De importancia especial es la encorvadura dorsal aguda del bronquio principal izquierdo en un nivel superior al de todos los orificios lobulares de ese lado.

2) La reformación del árbol izquierdo parece predisponer al desarrollo de complicaciones endobronquiales.

3) Nos parece que lo más cuerdo es tratar de obtener la elevación óptima del diafragma, basada en observaciones broncoscópicas del caso individual, aunque no sea la elevación máxima obtenible.

## RESUME

1) Les mesures de collapsothérapie qui produisent une élévation du diaphragme causent une déformation caractéristique des bronches principales. La courbure dorsale aigue de la bronche principale gauche au-dessus du niveau des orifices lobulaires est particulièrement significative.

2) La déformation de l'arbre gauche parait favoriser le développement des complications endo-bronchiques.

3) Il faut tenter d'obtenir l'élévation optimum du diaphragme, basée sur l'observation bronchoscopique du cas particulier, plutôt que l'élévation maximum.

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# Primary Bronchogenic Leiomyoma

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Jackson Heights, New York

The purpose of this paper is to report an unusual type of bronchial new growth discovered at operation in an elderly male. The patient presented a rather characteristic clinical picture; the symptoms, physical and radiologic findings suggested an occluding bronchogenic neoplasm of the right upper lobe. Since the tumor could not be visualized through the bronchoscope and no specimen for diagnosis could be obtained, an exploratory thoracotomy was performed. At operation, on gross examination, the differentiation between a benign and malignant lesion could not be made, but the conditions at the time necessitated a pneumonectomy. Histological examination of the tumor revealed a leiomyoma, primary in the bronchus. The patient made an uneventful recovery.

Randall and Blades[1] recently reviewed the literature on mesenchymal tumors of bronchial origin. They note that reports of such tumors are exceedingly scarce. In this category, chondromatous tumors are the most frequently reported. As to tumors arising from smooth muscle and limited to the bronchus, they found six such previously reported, of which four can be considered benign and two malignant. Among the benign tumors are leiomyoma reported by Franco,[2] Forkel,[3] and Duessing,[4] and fibroleiomyoma reported by Brahdy.[5] In the malignant category are Neumann's cancerous tumor of smooth muscle, presumably leiomyosarcoma[6] and Kramer's myoblastoma.[7] To this series, Randall and Blades add their case which reports a leiomyosarcoma in a 34 year old Negro, diagnosed by bronchoscopic biopsy. Our search of the literature revealed one additional case not cited in the Randall and Blades series which was reported by Brunn and Goldman.[8] This was a leiomyosarcoma of the left main stem bronchus. Thus the present case represents the ninth reported of a tumor arising from smooth bronchial muscle and only the fifth benign leiomyoma in this series.

Whether or not the scarcity of such case reports reflects truly an actual rare incidence of this type of tumor, or whether many are seen and not reported is a speculative matter. Jackson and Jackson,[9] in their extensive clinical experience, state that their records of benign bronchial tumors include myomas although they do not cite specific cases. On the other hand, one can appreciate

the low incidence of benign bronchial tumors in general as compared to malignant from a 15 year series of cases reported by Adams,[10] of which 157 are malignant and seven were benign—a ratio of 22 to one.

### *Case Report*

J. E., a 61 year old white male paper hanger was first seen on February 23, 1948, complaining of symptoms of a "common cold," viz, sore throat, running nose, and persistent cough with minimal expectoration of a heavy yellowish phlegm. Further questioning revealed that cough had been present for about eight months previously. There was no history of hemoptysis, chills, hyperpyrexia, night sweats, chest pain, dyspnea, orthopnea, peripheral edema or loss of weight.

Physical examination revealed a well-developed, well-nourished white male who looked younger than his years. No supraclavicular, axillary or intercostal nodes were palpable. Oral temperature was 100 degrees F. Blood pressure was 240 systolic, 120 diastolic. There was slight injection of the pharynx and nasal mucous membrane with a mucoid nasal discharge. Examination of the chest revealed dullness to percussion in the right subclavicular area and bronchovesicular breathing. Occasional sibilant rhonchi but no rales were heard over this same area.

Fluoroscopic inspection disclosed a sharply circumscribed triangular area of density extending from the upper pole of the right hilum and upward and outward to the chest wall. The trachea was deviated to the right. Other findings were essentially negative.

Roentgenography on March 4, 1948, was reported as follows: "There is a sharply circumscribed area of infiltration seen to extend from the upper pole of the right hilum to the chest wall. This fans out and becomes wider toward the periphery. It occupies the first anterior intercostal space. The trachea is sharply deviated to the right from the level of the clavicles to the second right rib. On lateral view this shadow appears to be anteriorly placed and occupies the upper third of the chest. Impression—segmental atelectasis of the right upper lobe."

Bronchoscopic examination on March 13, 1948, showed slight injection of the mucosa around the opening to the right upper lobe bronchus. All the bronchial orifices were patent and no evidence of endobronchial disease could be seen.

Sputum examination was negative for acid fast bacilli on concentrate and culture. Papanicoloau stain was negative for cancer cells. X-ray film repeated on March 15, 1948, showed no change but on April 7 (Figure 1) there was evidence of extension of atelectasis. He was referred for exploratory thoracotomy.

### *Surgical Report*

Patient was admitted to Beth David Hospital, New York City, on April 5, 1948. Exploratory thoracotomy was performed (AEB) on April 8, 1948, through an anterior approach. The right upper lobe was found to be completely atelectatic. A firm mass (Figure 2) about 3 x 4 cm. was felt which seemed to encircle the upper lobe bronchus. This mass was adherent to the middle lobe, obliterating the fissure. Many soft mediastinal glands were felt. The tissues bled freely. The patient's blood pressure was 240/160. It seemed that a pneumonectomy, based on the assumption that the mass was malignant, was the simplest and most promising procedure.

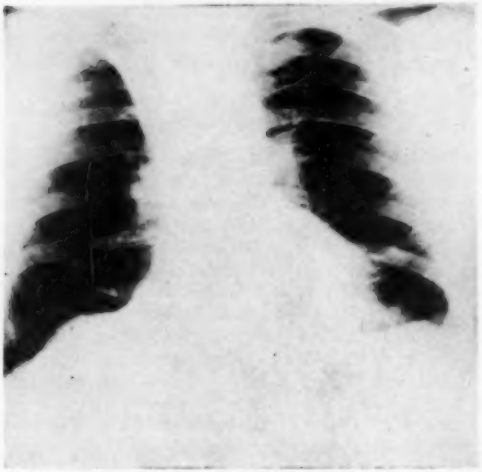

FIGURE 1: Chest x-ray film showing triangular area of atelectasis in right upper lobe.

FIGURE 2: Shows tumor in situ at opening of right upper lobe bronchus.

This was performed, using the individual ligation technique.

On opening the lung a nodule about 1.0 cm. in diameter was found occluding the right upper lobe bronchus about 0.5 cm. from the cut margin of the bronchial stump. The nodule was firm, encapsulated and covered with mucous membrane. It was pedunculated and attached to the posterior bronchial wall by a fine pedicle. On section the nodule was grayish white, and had a trabecular architecture. The right upper lobe showed areas of atelectasis and solidification.

*Pathologic Report*

Specimen consists of a right lung measuring 18 by 16 cm. in diameter. The lung is rather firm and little aerated. The pleura is thickened and shows numerous fibrous and many fibrinous adhesions. The interlobar fissure is partly obliterated. There is a tumor nodule near the entrance of the right upper lobe about 0.5 cm. distal from the bronchial stump. The nodule is pedunculated. It is firm, encapsulated and measures 1 cm. in diameter. On section it is greyish white in color and has a trabecular architecture. The surrounding bronchus is natural. On section the lung is congested and shows areas of atelectasis and solidification which are most marked in the upper lobe.

Histologically, sections of the tumor (Figures 3 and 4) showed it to be composed of generally elongated elements with vesicular nuclei which have blunted ends and are fairly uniform in size. The cellular outlines are indistinct. In several places it is possible to recognize intra-cellular longitudinal fibrils. The tumor extends up to immediately beneath the respiratory epithelium from which it is separated by a thickened and hyalinized basal membrane. With a Van Gieson stain the neoplastic elements stain yellow and show practically no collagen between the individual cells.

Microscopic diagnosis: Pedunculated subepithelial leiomyoma of the bronchus. Bronchial obstruction. Chronic pneumonitis. Patchy atelectasis.

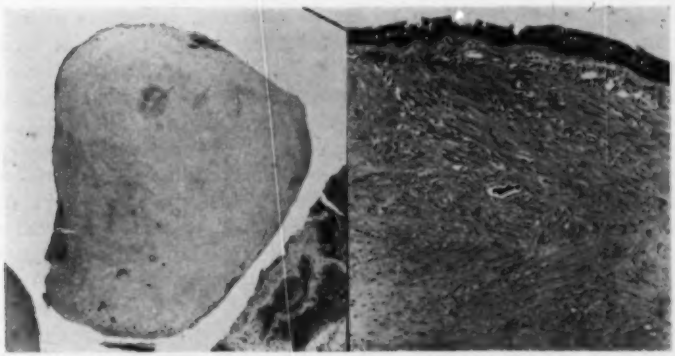

FIGURE 3 FIGURE 4

*Figure 3:* Very low power photomicrograph showing a portion of the tumor covered by respiratory epithelium.—*Figure 4:* High power photomicrograph showing that the tumor consists of interlacing bundles of well differentiated smooth muscle. The neoplastic tissue is separated from the respiratory epithelium by a thickened and hyalinized basal membrane.

## SUMMARY

An unusual tumor of mesenchymal tissue of bronchogenic origin is presented. This is the fifth of its kind reported in the literature.

## RESUMEN

Se presenta un raro tumor de tejido mesenquimatoso de origen broncógeno. Este es el quinto tumor de ese tipo comunicado en la literatura.

## RESUME

L'auteur rapporte un cas rare de tumeur de tissu mésenchymateux d'origine bronchogénique. C'est le 5ème cas relevé dans la littérature.


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# Editorial

## TUBERCULOSIS PROPHYLAXIS

In this issue of *Diseases of the Chest* Manoel de Abreu emphasizes the importance of early diagnosis and prompt treatment as the most important tuberculosis prophylactic measure known to the medical profession. Having long recognized this method plus isolation of contagious cases as the only truly effective one, Dr. de Abreu devised photofluorography and presented it to the world in 1936. The only difference between this and the screening procedure established by the Powers X-ray Corporation on recommendation of the Queensborough Tuberculosis and Health Association in New York which has been in use since 1931 was that Dr. de Abreu used miniature celluloid film, while the New York group employed 14 x 17 inch paper film. Both methods permit the exposure of approximately 1,000 chest per day by a single unit at much less expense than by any method previously introduced. These rapid methods of making x-ray inspections of the chest represented one of the finest contributions to the control of tuberculosis of all time. They quickly screen from any group the bearers of shadow-casting chest lesions.

While these shadows do not constitute diagnoses, they denote the presence and location of gross disease, the etiology of which can often be determined by specific tests, microscopical and bacteriological studies. Many such lesions prove to be tuberculous. If their hosts can promptly be placed under whatever management is necessary or indicated to prevent the spread of their tubercle bacilli to others, relatively few of the members of the oncoming generation become infected with tubercle bacilli. The time arrives when the most refined method is applicable, namely, the administration of the tuberculin test to all and seeking the sources of infection among the reactors' associates and keeping all reactors under close observation in order to find clinical disease which they may develop before it becomes contagious. This method will lead to the eradication of tuberculosis in any area where it is conscientiously practiced. Indeed, this is the only known solution of the tuberculosis problem in the world. Thus the procedure that Dr. de Abreu is employing and strongly recommending for Brazil and all other parts of the world where tuberculosis is prevalent is fundamental and sure to bring results wherever the people have the will to free themselves from this disease.

Dr. de Abreu especially emphasizes the importance of mass chest x-ray surveys in the prophylaxis of tuberculosis. He states

that in 1937 only 20,000 such x-ray inspections of the chest were made in Brazil; in 1945, 150,000, and in 1949, 1,500,000. In the United States he points out that mass x-ray surveys reached a million people in 1945 and 14,000,000 in 1949. This method is also now being widely used in other nations.

There has been a marked decrease in tuberculosis mortality in Rio de Janeiro since 1945. That year it was 342 per 100,000 and in 1949 it was only 242, a decrease of 29.2 per cent. This has been most noticeable between the ages of 20 and 40 years. Dr. de Abreu believes this rapid decrease is the result of progressive use of photofluorography leading to early diagnosis and prompt treatment.

While a definite decrease has occurred in tuberculosis mortality among adults in Rio de Janeiro, a significant increase in mortality has occurred among children from birth to five years. De Abreu points out that this increase in mortality among children does not show a favorable influence of the large and progressive use of BCG among them.

Apparently only two new factors have been introduced in the Rio de Janeiro tuberculosis situation in recent years, namely, photofluorography and BCG. The former applied to adults, the latter largely to infants and children. Among infants from birth to one year the mortality rate was 46 per 100,000 in 1945, and 65 in 1948, an increase of 41.3 per cent. Among children from one to two years the increase was 62.5 per cent between 1945 and 1948, and 15.9 per cent among those from two to four years of age.

In New York City where BCG was not introduced except in one extremely small study, the tuberculosis mortality rate among infants from birth to one year was only 11 per 100,000, and among male and female children from birth to four years it was 8 and 11 respectively in 1948. Between 1915 and 1948 the mortality rate among infants changed from 197 to 11 per 100,000, a decrease of 94 per cent. During the same years a 95 per cent decrease occurred among boys, with an 83 per cent decrease among girls from birth to four years.

With no other factor than BCG having been introduced among children under five years of age in Rio, questions immediately arise, not only with reference to inefficaciousness of BCG, but also as to whether directly or indirectly it may have been harmful. Have the parents of young children developed such a sense of security by having been told that BCG immunizes that they have disregarded hygienic measures to protect them against virulent tubercle bacilli? Has BCG regained its virulence and actually been responsible for at least a part of this increase in mortality? This report of increased mortality among young children of Rio immediately following those of Vorwald of Saranac Lake and Dubos

of the Rockefeller Institute to the effect that silicotic guinea pigs and mice on deficient diets develop progressive and killing tuberculosis from BCG as well as a number of reports in recent years showing that BCG markedly changes its virulence in culture medium should cause everyone to demand that many facts which we do not now possess be established concerning this organism.

Tubercle bacilli, virulent or avirulent, introduced into the human body by any route including the digestive and respiratory tracts, subcutaneous tissue, or within the layers of the skin are promptly phagocytosed in considerable numbers and carried by neutrophils to various parts of the body. Lemon and Feldman placed tubercle bacilli into the pleural cavities of animals and found that within a short time they had been focalized not only in the lungs, but in numerous other remote parts. Krause injected tubercle bacilli subcutaneously into the groins of small animals. Within four days he found them in the lungs and tracheobronchial lymph nodes. Vorwald and Long injected tubercle bacilli directly into the blood stream of animals and soon found them focalized in the lungs.

As early as 1890 Dobrokowski demonstrated that tubercle bacilli in the digestive tract of guinea pigs pass through the intestinal walls without producing demonstrable lesions. Five years later, Desoubry showed that bacteria are carried through the intestinal wall of dogs during the digestion of fat so that they can be detected in the chyle on plate culture. Cotton, Mohler, Ravenel, Schroeder and others have introduced tubercle bacilli into the digestive tract and within a few hours after entering the stomach they were found in abundance in the contents of the thoracic duct.

For the most part infants and children of Brazil have received BCG by the oral route. During the first few days of life three doses of 30 milligrams each have been administered at two or three day intervals. These organisms are promptly distributed by the blood stream to various parts of the body, including the lungs. This is essentially Calmette's original method.

If BCG is introduced intradermally it results in lesions in at least the innermost layers of the skin and in the regional lymph nodes. From these points bacilli may be carried by phagocytes to numerous parts of the body.

Dubos has emphasized the importance of the physiological state of animals with reference to the development of clinical and killing tuberculosis from BCG. The physiological state of human beings, particularly infants, children and young adults, may be equally important. He says: "It is even conceivable that many of the classes of human beings who are most dangerously exposed to tuberculosis (for example those living under conditions of great economic stress with inadequate food supplies) may be those least

capable of responding satisfactorily to immunization procedures." Dietary deficiencies are not uncommon; in fact, they are almost the rule in some parts of the world and may be present in children thought to be "well fed." Moreover, physiological status of humans may change in a brief period of time.

Dubos points out that there is much reason to suspect that many of the controversies elicited by BCG vaccination cannot be settled until a number of bacteriological and immunological problems have been solved.

The reports of Vorwald and Dubos regarding silicotic animals and those on deficient diets developing progressive and killing tuberculosis from BCG, the increase in tuberculosis mortality among children under five years of age in Rio de Janeiro where BCG has been employed and marked increase in virulence which BCG attains on certain artificial media may well be regarded as warning signals. In any event, they suggest that this is a good time to pause not only with reference to so-called mass vaccination but also that of "special groups" and refer BCG to bacteriologists and immunologists such as Dubos for impartial studies leading to the establishment of facts concerning BCG of which we have almost none at this moment. While this is being accomplished, the medical profession would later have no regret if it now strictly limits its support of BCG to the studies in progress by the Division of Tuberculosis of the United States Public Health Service.

J.A.M.

# The New Home of the College


*On July 1, 1951, the Executive Offices will occupy this building recently purchased by the American College of Chest Physicians. Located at 112 East Chestnut Street, it is one-half block from Michigan Boulevard, along Chicago's "Magnificent Mile," and near the Drake Hotel and the Palmolive Building, famous Chicago landmarks.*

# SEVENTEENTH ANNUAL MEETING OF THE COLLEGE

## Ambassador Hotel, Atlantic City, New Jersey

## JUNE 7 THROUGH 10, 1951

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*One Hundredth Annual Session*
**AMERICAN MEDICAL ASSOCIATION**
**Atlantic City, June 11 - 15, 1951**

The 1951 Annual Meeting of the College will be held in Atlantic City, New Jersey, at the Ambassador Hotel, where so many successful meetings of the College have been held. It is both interesting and gratifying to report that advance hotel reservations have already exceeded all previous records. In order not to be disappointed, members are urged to make their reservations at once by writing directly to the Ambassador Hotel.

The Committee on Scientific Program of the College, under the chairmanship of Dr. Edwin R. Levine of Chicago, has released the following preliminary program:

### *Preliminary Program*

**Exploratory Surgery of the Heart,**
Charles P. Bailey, Philadelphia, Pennsylvania.

**Title to be announced,**
Ignacio Chavez, Mexico City, Mexico.

**Bronchiectasis in Children,**
Lloyd B. Dickey, San Francisco, California.

**Venous Pressure as a Guide to Pneumoperitoneum Therapy in Pulmonary Emphysema,**
Chesmore Eastlake and Gustav J. Beck, New York, New York.

**Relation of X-ray Survey to Bronchogenic Carcinoma (Committee Report),**
Alfred Goldman, Beverly Hills, California, Chairman.

**Studies in Pericarditis,**
George R. Herrmann, Galveston, Texas.

**The Status of Tried Procedures in the Treatment of Pulmonary Tuberculosis as Seen in the Light of Thirty Years Experience,**
William A. Hudson and Willard B. Howes, Detroit, Michigan.

**The Relationship Between Pathological Changes in Blood Vessels in Resected Lobes and Lungs as Correlated with Pulmonary Artery Pressure Changes Recorded During Cardiac Catheterization,**
Allan Hurst, Denver, Colorado.

**Recovery of Pulmonary Function After Crushing Injuries of the Chest,**
N. K. Jensen, Minneapolis, Minnesota.

**A Study of Delayed Non-Specific Tuberculin Reactions,**
M. R. Lichtenstein and Carmine Cilella, Chicago, Illinois.

**Physiologic Factors in the Use of the Respirator for Impairment of Respiratory Function,**
J. V. Maloney, Jr., Baltimore, Maryland.

**The Effect of the Diminishing Incidence of Primary Infection on the Tuberculosis Control Program,**
Jay Arthur Myers, Minneapolis, Minnesota.

**Respiratory Air Flow and Related Measurements in Pulmonary Disease,**
Donald F. Proctor, Baltimore, Maryland.

**The Application of Pancreatic Enzyme in the Treatment of Tuberculous Empyema,**
Louis C. Roettig and Louis Mark, Columbus, Ohio.

**Relationship of the Autonomic Nervous System and Peripheral Vascular Bed to the Acute Pulmonary Edema State,**
Stanley J. Sarnoff, Boston, Massachusetts.

**Clinical Diagnosis of Pseudo-Truncus Arteriosus,**
Helen B. Taussig, Baltimore, Maryland.

**Follow-up of Pneumothorax (Committee Report),**
Harold G. Trimble, Oakland, California, Chairman.

**The Recognition of Non-allergic Asthma,**
Leon Unger, Chicago, Illinois.

**Current Concepts on the Etiology of the Common Cold,**
Thomas G. Ward, Baltimore, Maryland.

**Pectus Excavatum,**
Francis M. Woods, Richard H. Overholt and H. E. Bolton, Boston, Massachusetts.

**Symposium on Chemotherapy and Antibiotics,**
Karl H. Pfuetze, Chicago, Illinois, Moderator.

**Symposium on Industrial Diseases,**
Edward C. Holmblad, Chicago, Illinois, Moderator.
(This symposium is being arranged with the cooperation of the American Association of Industrial Physicians and Surgeons).

**Symposium on ACTH and Cortisone,**
Moderator to be announced.

**X-Ray Conference,**
Leo G. Rigler, Minneapolis, Minnesota, Moderator.

**Motion Picture Session,**
Paul H. Holinger, Chicago, Illinois, Chairman.

**Round Table Luncheons,**
Harold G. Trimble, Oakland, California, Chairman.

The program has been arranged to provide ample time for discussion from the floor. Members are invited to bring slides and other illustrative material for discussion of papers. During the symposiums, members may make comments from the floor, as well as ask questions.

ANNUAL MEETING, ARGENTINE CHAPTER, MAR DEL PLATA, NOVEMBER 25, 1950

Seated, from left to right: Dr. Jose Peroncini, Dr. Bolognesi, Dr. Cuchiani, Dr. Pezzati, Dr. Sayago, Dr. Oscar Vaccarezza, Dr. Raul F. Vaccarezza, Dr. Rojas and Dr. Lopez Bonilla.

# College Chapter News

## CALIFORNIA CHAPTER

The annual meeting of the California Chapter of the College will be held at the Ambassador Hotel, Los Angeles on May 12. The following program will be presented:

***Morning Session:***
J. J. Singer, Moderator.

"Cortisone and ACTH in Tuberculosis,"
David Salkin, Sol Netzer and Emil Bogen.

"BCG Vaccination in Pulmonary Sarcoidosis,"
Marvin S. Harris.

"The Clinical Application of the Warring Test for Pulmonary Ventilatory Function,"
Harold Guyon Trimble and James R. Wood.

"Radiological Findings in Early Carcinoma of the Lung,"
Eugene Freedman and James H. Billings.

"Treatment of Abscesses Associated with Spinal Tuberculosis,"
John H. Aldes.

"The Management of Esophageal Perforations,"
David J. Dugan.

"The Surgical Treatment of Certain Asymptomatic Intrathoracic Lesions Discovered by Roentgen Examination,"
Lyman A. Brewer, III.

"The Repair of Experimental Tracheal Defects with Fresh and Preserved Homologous Tracheal Grafts,"
Orland G. Davies, J. Malcolm Edmiston and H. J. McCorkle.

"Extracorporeal Oxygenation of Blood,"
Joseph H. Miller.

***Luncheon:***
Seymour M. Farber, Moderator.

*Guest Speaker:* Charles P. Bailey, Philadelphia, Pennsylvania, "Pulmonary Resection."

***Afternoon Session:***
Lyman A. Brewer, III, Moderator.

"Diagnosis of Congenital Heart Disease,"
William Paul Thompson.

"Cardiac Catheterization in Surgical Heart Disease,"
Sidney S. Sobin and Louis E. Martin.

"Transposition of Pulmonary Veins: Report of Five Cases Diagnosed by Cardiac Catheterization,"
David C. Levinson, R. S. Cosby, Willard Zinn, Sima P. Dimitroff, Robert Oblath, Telfa Reynolds and George C. Griffith.

"Auricular Paroxysmal Tachycardia in Man During Thoracic Surgery,"
Eliot Corday, Alfred Goldman, Robert W. Oblath and Myron Prinzmetal.

"Intracardiac Digital Surgery,"
Charles P. Bailey, Robert P. Glover, Thomas J. E. O'Neill and Marvin M. Lacy.

"Coarctation of the Aorta,"
Bert Meyer and John C. Jones.

"The Surgical Treatment of Valvular Pulmonic Stenosis,"
William H. Muller, Jr.

Cocktails will be served at 5:00 p. m., after the close of the scientific session.

C. Gerald Scarborough, *Secretary*

Alfred Goldman, Chairman *Program Committee*

Louis I. Sokol, Chairman *Arrangements Committee*

**POSTGRADUATE COURSE IN DISEASES OF THE CHEST, HAVANA CUBA, DECEMBER 18-22, 1950.**

Some of the physicians and instructors who participated in the postgraduate course in Diseases of the Chest sponsored by the Cuban Chapter of the College.

## CUBAN CHAPTER

The Cuban Chapter of the College sponsored a postgraduate course in diseases of the chest during the week of December 18-22, 1950, in celebration of the Tenth Anniversary of the chapter. The course was held at the Instituto de Cirugia Ortopedica in Vedado, Havana. Seventy-five physicians attended the postgraduate course; a photograph of some of the registrants and lecturers appears on page 368. Dr. Pedro Farinas, President of the Cuban Chapter, served as director of the postgraduate course committee and Dr. R. Sanchez Acosta, Secretary-Treasurer of the chapter, served as secretary. Other members of the postgraduate course committee were Dr. Frank Barrera, program, Dr. R. Gomez Zaldivar, scientific exhibits, Dr. Arnaldo Coro, commercial exhibits, Dr. Ramon Casas, reception, and Sr. L. Rodriguez Machin, publicity. Dr. Chevalier L. Jackson, Philadelphia, Pennsylvania, President-Elect of the College, attended the course on the opening day and extended the best wishes of the national organization at a luncheon meeting. The course closed with a banquet held at the Circulo Medico de Cuba which was attended by more than 150 physicians. Guests of honor were the Minister of Health of Cuba and the President of the Cuban Medical Association. The Cuban Medical Association was very cooperative with the chapter in the preparation and presentation of the postgraduate course.

A special meeting of the Cuban Chapter was held on February 5th at the Curie Hospital in Havana. Dr. Edgar Mayer of New York City was guest speaker at the meeting. The following program was presented:

"Report to the Chapter on the First International Congress on Diseases of the Chest, Rome, Italy, September 17-22, 1950,"
Antonio Navarrete, Regent for Cuba and President of the Cuban Delegation to the Congress.

"Pulmonary Fibrosis and Emphysema with Special Reference to Dust Inhalation,"
Edgar Mayer, New York, New York.

"Presentation of Cases,"
Ricardo Sánchez, Acosta.
Dr. Pedro Farinas, President of the Chapter, presided at the meeting.

R. Sánchez Acosta, Secretary.

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## ILLINOIS CHAPTER

The Illinois Chapter of the College will hold its annual meeting at the Sherman Hotel, Chicago, on Wednesday, May 23, at the time of the annual session of the Illinois State Medical Society.

William J. Bryan, Secretary.

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## OHIO CHAPTER

The Ohio Chapter of the College will meet at the Netherland Plaza Hotel, Cincinnati, on April 25, at the time of the Ohio State Medical Association meeting. Dr. Harold I. Humphrey of Cincinnati will be guest speaker at the chapter meeting. His subject will be "The Treatment of 63 Tuberculosis Patients with Thiosemicarbasone (Tibione and Pabtec)."

Harold G. Curtis, *Secretary*.

## FLORIDA CHAPTER

The Third Annual Meeting of the Florida Chapter of the College will be held at the Hollywood Beach Hotel, Hollywood, Florida on April 22. The session will open with a business meeting at 10:00 a. m. followed by a scientific program.

"Nontuberculous Pulmonary Pathology Detected on X-ray Surveys,"
Clarence M. Sharp, Jacksonville.

"Antibiotics in the Treatment of Tuberculosis,"
Henry C. Sweany, Jacksonville.

"Classification and Diagnosis of Mediastinal Tumors,"
Hawley H. Seiler, Orlando.

"Hemoptysis of Undetermined Origin,"
Nathaniel M. Levin, Miami.

"Congenital Cysts of the Lung,"
DeWitt C. Daughtry, Miami.

***Luncheon Meeting:***

"Cancer of the Lung,"
Jack Reiss and Harry Baum, Miami; and
Maurice Kovnat, Miami Beach.

X-Ray Round Table Discussion,
M. Jay Flipse, Miami, Moderator.

Alexander Libow,
*Secretary*

Nathaniel M. Levin, Chairman
*Program Committee*

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## MINNESOTA CHAPTER

The Minnesota Chapter of the College will hold its annual meeting in Rochester on April 30, at which time the following program will be presented:

"Coin Lesions of the Lung,"
Daniel L. Fink, Minneapolis.

"Crushing Injuries of the Chest,"
Nathan K. Jensen, Minneapolis.

"Bronchial Asthma,"
Royal V. Sherman, Red Wing.

"The Association of Pulmonary Tuberculosis and Bronchogenic Carcinoma,"
G. A. Hedberg, Nopeming,
H. Graham and W. H. Wierman, Rochester.

"Surgery for Diseases of the Heart and Great Vessels,"
J. W. Kirklin, Rochester.

Arthur M. Olsen, Chairman, *Program Committee.*

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## MISSOURI CHAPTER

The annual meeting of the Missouri Chapter of the College will take place at the President Hotel, Kansas City, on Sunday afternoon, April 22 at 1:00 p. m. The following program will be presented:

***12:30 p.m.:*** **Meeting of the members of the Missouri Chapter.**

***1:00 p.m.:*** **Luncheon.**

***Scientific Program:***

"The Sinuses and Bronchiectasis,"
G. O. Proud, Kansas City, Kansas.

"The Therapeutic Potentialities of Artificial Pneumoperitoneum,"
Andrew L. Banyai, Milwaukee, Wisconsin.

"The Diagnosis and Management of Circumscribed Lesions of the Lungs,"
Corrin H. Hodgson, Rochester, Minnesota.

X-Ray Conference,
Lawrence E. Wood, Kansas City, Kansas, Moderator.

It is suggested that members of the Missouri Chapter send their interesting x-ray films to Dr. Wood for consideration. An interesting series of films will be selected for presentation in the x-ray conference. Please send films and case reports to Dr. Lawrence E. Wood, Department of Medicine, University of Kansas Medical School, Kansas City, Missouri.

Alexander J. Steiner,
*Secretary*

J. S. Hoffman, Chairman
*Program Committee*

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## NEW YORK STATE CHAPTER

The annual meeting of the New York State Chapter will be held at the Statler Hotel, Buffalo, on May 3. Dr. Charles P. Bailey of Philadelphia will be the guest speaker at the evening dinner meeting. A program arranged by Dr. Donald R. McKay, Buffalo and Dr. David Ulmar, New York City, will be presented in the section on diseases of the chest of the New York State Medical Society on Thursday afternoon, May 3, and Friday morning, May 4.

Harry Golembe, *Secretary.*

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## VIRGINIA CHAPTER

The Virginia Chapter of the College will meet at the Richmond Academy of Medicine Building on April 25. The scientific program will start at 10:30 a. m. and continue throughout the afternoon.

W. E. Roye, Chairman, *Program Committee.*

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## TEXAS CHAPTER

The Texas Chapter of the College will hold its annual meeting at the Buccaneer Hotel in Galveston on Monday, April 30. The registration and scientific program will open at 9:00 a. m. The following program will be presented:

"Surgical Treatment of Emphysematous Bullae,"
Howard T. Barkeley, Houston.

"Medical Management of Pulmonary Emphysema,"
Daniel E. Jenkins, Houston.

"Respiratory Physiology,"
William F. Miller, Dallas.

X-Ray Conference,
Martin Schneider, Galveston.

***Luncheon Meeting:***
Business Session and Election of Officers.

"Surgery for Stenotic Valvular Disease of the Heart,"
Clive R. Johnson, Fort Worth.

"Thoracoplasty and Resection in the Treatment of Pulmonary Tuberculosis,"
Robert R. Shaw, Dallas.

"Clinical Pathologic Conference,"
Paul Brindley, Galveston.

Henry R. Hoskins,
*Secretary*

John W. Middleton, Chairman
*Program Committee*

# College News Notes

Dr. Karl Pfuetze, formerly medical director and superintendent of the Mineral Springs Sanatorium, Cannon Falls, Minnesota, has accepted the position as medical director and superintendent of the new 500-bed tuberculosis hospital now under construction at the University of Illinois Medical Center in Chicago.

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Consolidation of the functions of the Division of Tuberculosis and the Division of Chronic Disease in the Public Health Service has been announced. The functions of these divisions will be carried on in the new division of Chronc Disease and Tuberculosis. It will be headed by Dr. Robert J. Anderson who has been chief of the Division of Tuberculosis for the past two and a half years. Dr. Anderson serves as Governor of the College for the U. S. Public Health Service.

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Dr. C. C. Howard, Glasgow, Kentucky, President of the Southeastern Surgical Congress, will present a paper on the subject of "Accidents and a Proposed Program for Prevention" at the Congress to be held in Hollywood, Florida, April 11-14. Dr. Otto C. Brantigan, Baltimore, Maryland, will present a paper on "Thoracoplasty in the Treatment of Pulmonary Tuberculosis" at the Congress.

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The Pollak Hospital, the Tuberculosis Unit of Peoria State Hospital, Illinois, was dedicated on December 3, 1950, in honor of Dr. Maxim Pollak, former superintendent of Peoria Municipal Tuberculosis Sanitarium and a member of the Tuberculosis Advisory Committee of the Illinois Department of Public Welfare.

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Dr. Edward A. Piszcek, Oak Forest, has been promoted from assistant professor to associate professor of public health at the University of Illinois College of Medicine.

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**COLLEGE EVENTS**

**17th Annual Meeting, American College of Chest Physicians, Atlantic City, New Jersey, June 7-10, 1951.**

Philadelphia Postgraduate Course, Philadelphia, March 26-30, 1951.
Milwaukee Postgraduate Course for General Practitioners, Milwaukee, Wisconsin, April, 1951.
Annual Meeting, Missouri Chapter, Kansas City, April 22, 1951.
Annual Meeting, Virginia Chapter, Richmond, Va., April 25, 1951.
Annual Meeting, Minnesota Chapter, Rochester, April 30, 1951.
Annual Meeting, Texas Chapter, Galveston, April 30, 1951.
Annual Meeting, New York State Chapter, Buffalo, May 3, 1951.
Annual Meeting, California Chapter, Los Angeles, May 12, 1951.
Annual Meeting, Illinois Chapter, Chicago, Illinois, May 23, 1951.
Chicago Postgraduate Course, Chicago, Ill., September 24-28, 1951.
Minneapolis Postgraduate Course, Minneapolis, October 18-20, 1951.
New York Postgraduate Course, New York City, November 12-17, 1951.

## MEDICAL SERVICE BUREAU

### POSITIONS AVAILABLE

Resident on service approved for training in pulmonary diseases, 500-bed county hospital with 130 sanatorium beds; large chest clinic. Recent graduate, one year residency or equivalent, preferred. Opportunity for surgical experience. Salary $235 monthly, increased to $450 September 1, 1951. Meals and laundry. Contact Director, Santa Clara County Sanatorium, San Jose, California.

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Staff physician wanted for a tuberculosis hospital in Southern California. Full medical and surgical care. Excellent opportunity for beginners to learn pulmonary diseases and for those with experience, a desirable staff position. Good salary. Please write Box 218A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

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### POSITIONS WANTED

Physician, experienced, draft exempt, wants position as medical director of small, modern tuberculosis hospital. Fellow, American College of Chest Physicians. Please address Box 248B, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

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Experienced chest physician desires position in tuberculosis sanatorium. Educated and trained in Europe. Has first citizenship papers and is licensed to practice medicine in State of New York. References concerning experience and list of publications available. Please address Box 249B, American College of Chest Physicians, 500 N. Dearborn St., Chicago 10, Ill.

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Position wanted as administrator of hospital or sanatorium. Experience. References available. Please address Box 250B, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

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Thoracic surgeon, 36 years of age, with considerable experience in surgical treatment of tuberculosis, qualified for boards in thoracic and general surgery, now engaged in private practice, desires position doing primarily or solely thoracic surgery. In draft classification IV. Will accept group, associate, or sanatorium position. Please address Box 251B, American College of Chest Physicians, 500 N. Dearborn St., Chicago 10, Illinois.

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Especially well trained and qualified tuberculosis specialist, specialty board in internal medicine, sub-specialty tuberculosis since 1934; associated with Forlanini Institute, Rome, many years; 16 years experience in pulmonary tuberculosis and chest diseases; 10 years medical director and superintendent of tuberculosis sanatoria, also chief of tuberculosis control program. Licensed in New York State, 42 years of age, excellent health, immediately available. Would accept responsible position commensurate with qualifications. Please address Box 253B, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

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The American Sterilizer Company has announced the appointment of Mr. Adrian Comper as general sales manager. Mr. Comper's sales department consists of fifteen different offices in metropolitan cities throughout the nation, manned by more than a hundred people, plus dealer representation in Canada and exclusive distribution by General Electric Medical Products Company outside the continent of North America.

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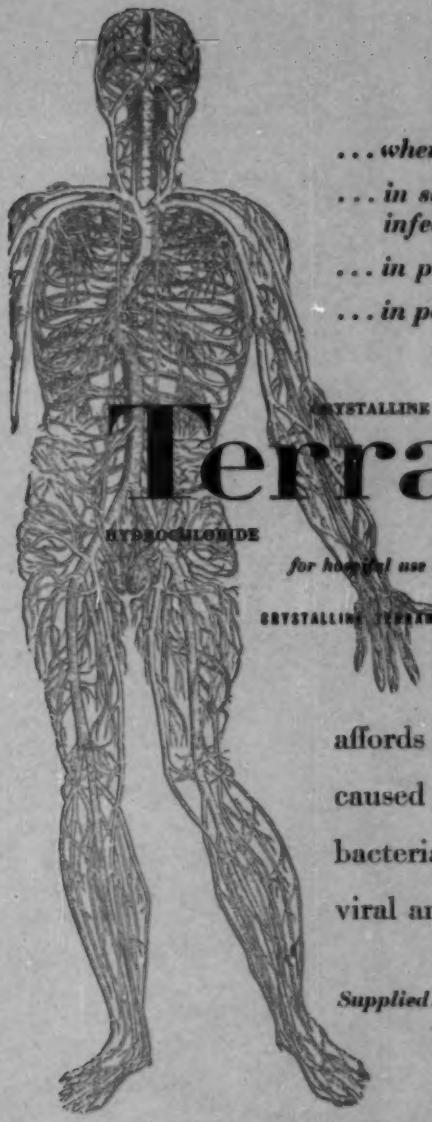

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